THE ENVIRONMENTAL FATE OF SELECTED POLYNUCLEAR AROMATIC HYDROCARBONS

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THE ENVIRONMENTAL FATE OF SELECTED POLYNUCLEAR AROMATIC HYDROCARBONS

by

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ABSTRACT

A review of the recent literature on polynuclear (polycyclic) aromatic hydrocarbons (PAH) has been carried out by SRI for general information on PAH and specific details about six selected PAH. The sources, transport, chemical and physical transformations, structure-reactivity relationships, and biological (non-carcinogenic) properties have been reviewed with recommendations for further research.

This review covers the literature through June 1975 with a few later references.

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TABLES

I INTRODUCTION

The Office of Toxic Substances (OTS), U.S. Environmental Protection Agency (EPA) under Contract No. 68-01-2681 requested that a literature search and evaluation of the results be undertaken for the following six polynuclear aromatic hydrocarbons (PAH): anthracene, benz(a)anthracene, benzo(a)pyrene, chrysene, 3-methylcholanthrene, and phenanthrene.

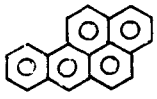
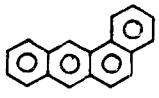
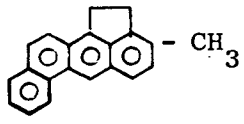
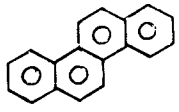
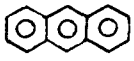
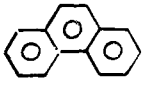
These six PAH are exemplary of the range of physical, chemical, and biological properties encountered among the several hundred known PAH. Since many, if not most, laboratory studies have involved two or more PAH, and naturally occurring PAH are usually complex mixtures containing up to several thousand compounds, as a practical matter we have organized this review by properties and reactivity rather than by individual compounds. Within each category, however, we have, whenever possible, emphasized specific structural features and structure-reactivity relationships. In many cases, comparisons among PAH involve compounds other than the six selected for this review and, where useful for our purposes, we have included them in our tabulations and discussion.

Since chemical nomenclature has undergone many (and often extreme) revisions in the last few years, Table 1 lists the common names of the PAH being studied, synonyms, and names used in current (1975) chemical literature.

Much of the information on the toxicity, accumulation, and degradation of polycyclic (polynuclear) aromatic hydrocarbons in the biological systems has been discussed and summarized in a comprehensive report published by the National Academy of Sciences (1972). This report attempts to provide supplementary information on the environmental fate

Table 1

NAMES AND SYNONYMS FOR SIX PAH

Common Name	Synonyms	1975 C. A. Nomenclature & No.
Benzo(a)pyrene 	3,4-Benzpyrene BP BaP 3,4-Benzopyrene	Benzo[a]pyrene 50-32-8
Benzo(a)anthracene 	1,2-Benzanthracene Benzanthrene Benzo(b)phenanthrene 2,3-Benzophenanthrene Tetraphene 1,2-Benz(a)anthracene	Benzo[a]anthracene 56-55-3
Methylcholanthrene 	Cholanthrene, 3-methyl- 20-MC MC 3-MC 20-Methylcholanthrene 3-Methylcholanthrene	Benz[j]aceanthrylene, 1,2-Dihydro-3-methyl- 56-49-5
Chrysene 	1,2-Benzphenanthrene	Chrysene 218-01-9
Anthracene 	Paranaphthalene	Anthracene 120-12-7
Phenanthrene 	None	Phenanthrene 85-01-8

and ecological effects of selected PAH. Carcinogenicity of the compounds has been specifically deleted. The reader is advised to see IARC Monograph, Volume 3.

For this study, OTS hopes to find answers to questions such as:

(1) Do tricyclic, tetracyclic, and pentacyclic aromatics react in the same way in the biosphere, and what is the principal mode of degradation?

(2) How does the degree of alkylation of the ring compound influence the mode of degradation?

(3) What are the degradation products, and are the fate and effects of these compounds known?

(4) How far up the food chain does bioaccumulation occur?

(5) How widespread is the metabolism of these compounds? Are they metabolized only by specialized organisms? Are only the non-alkylated compounds metabolized?

(6) Are the physical properties (solubility, volatility, etc.) such that they favor the conditions leading to degradation of these compounds?

The principal contributors to this report by area are:

Literature Search	S. B. Radding
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II SUMMARY, CONCLUSIONS, AND RECOMMENDATIONS

Summary and Conclusions

Table 2 summarizes our findings. Many simple, naturally occurring organic compounds can be readily pyrolyzed to complex mixtures of PAH at temperatures above 300°, with maximum yields at 700-900°C. Newer analytical techniques indicate that PAH found in the environment can be extremely complex mixtures containing up to several thousand components, including many alkylated PAH. Although PAH from natural combustion sources may differ significantly in structure from PAH from anthropogenic sources, as has been claimed, the evidence for this distinction appears to be equivocal; much more extensive data would seem to be required to decide the issue.

PAH are widely distributed in the environment. They are found in living animal and plant tissue, sediments, soils, air, and surface waters. Most PAH probably arise as pyrolysis products formed during combustion or heating of fossil fuels and of most natural products. The compounds may be natural products of animal and vegetable metabolisms, and are probably released from exposed fossil fuel deposits by erosion. PAH are essentially not soluble in water and have low vapor pressures, so that the major environmental transport mode is as particulate in air or water. However, comparison of PAH levels in plant and animal tissue suggests that concentration effects are not large despite large partition coefficients reflecting high solubility in fatty tissues. Presently used methods of analysis for estimating airborne concentrations of PAH may seriously underestimate the concentrations of some relatively volatile PAH such as pyrene, anthracene and benzo(a)anthracene.

Table 2

ENVIRONMENTAL DATA FOR SELECTED PAH[a]

	Anthracene	Benz(a)-anthracene	Benzo(a)-pyrene	Chrysene	3-Methyl-cholanthrene	Phenanthrene	Other PAH, Comments	Report Page Reference[b]
Physical Properties	4	4	4	4	3	4	All PAH have low vapor pressure and strong absorptivity to minerals and carbon; low solubility in water; all strongly absorb light in solar region and dissolve in fatty solvents. Some data on 30 PAH.	12
Vapor Pressure	3	3	3	1	1	3		18
Vaporization Rate	1	1	1	1	1	1		20
Adsorption	1	1	1	1	1	1		17
Solubility	3	2	2	1	1	3		17,19
UV Spectra	4	4	4	4	4	4		21
Partition Coefficient	3	3	3	3	3	3		11,12
Chemical Reactivity	4	3	3	2	1	4	Most PAH react rapidly with O_2 by self sensitized process to form quinones and other products. Ozone (or HO) will also oxidize PAH quickly. Alkylation accelerates these reactions but little specific data. Data on oxidation of 13 PAH. Quantitative estimates of half-lives for all PAH give 5 ± 5 hrs in environment.	3,24
RO_2 Radical	3	1	3	1	1	3		25-27
Singlet O_2	3	3	3	1	1	2		22-30,31,32,33
O_3 and Cl_2	4	3	3	2	1	3		34,41 36-38,40,41
HO Radical	1	1	1	1	0	1		39
Peracids	4	1	1	1	0	4		42
NO_x and SO_2	2	1	2	0	0	1		41,42
Sources							Both natural and anthropogenic combustion sources are major producers of many PAH. No firm basis exists for distinguishing the source based on structure of PAH. B(a)P most often used as a measure of occurrence of other PAH.	
Natural Fires	2	2	2	2	2	2		22
Coal Combustion	2	3	3	2	2	2		86,87
Incineration	2	3	3	2	2	2		90,92
Fuel Combustion	2	3	3	3	2	2		93
Industrial Processes	1	1	3	1	1	1		88,89
Seasonal	1	3	3	3	1	1		70,71
Forms	3	3	3	1	1	3		17,19,86

Table 2
(Continued)

ENVIRONMENTAL DATA FOR SELECTED PAH

	Anthracene	Benz(a)-anthracene	Benzo(a)-pyrene	Chrysene	3-Methyl-cholanthrene	Phenanthrene	Other PAH, Comments	Report Page Reference
Bioaccumulation								
Bacteria	2	2	4	0	2	2	9,10-dimethyl-1,2-benzanthracene, 1,2,5,6-dibenzanthracene, 1,2-benz-anthracene, 1,2-benzopyrene, pyrene, 1,12-benzperylene, perylene. See list on p. 55. (2)[a]	53,54,55
Algae	0	0	2	0	0	0	Perylene. (2)[a]	57
Higher Plants	2	2	4	2	0	2	See list on p. 57. (2)[a]	56
Invertebrates	0	0	4	0	0	0	Perylene. (2)[a]	57,58
Fish	0	0	2	0	0	0		
Amphibians	0	0	0	0	0	0		
Reptiles	0	0	0	0	0	0		
Birds	0	0	3	0	0	0		59
Mammals	0	0	3	0	3	0	1,2,3-dibenzanthracene, 1,2-benz-pyrene. (3)[a]	59
Biodegradation/Metabolism[c]								
Bacteria	3	0	0	0	0	3		60,61
Algae	0	0	0	0	0	0		
Higher Plants	0	0	0	0	0	0		
Invertebrates	0	0	0	0	0	0		
Fish	0	0	0	0	0	0		
Amphibians	0	0	0	0	0	0		
Reptiles	0	0	0	0	0	0		
Birds	0	0	0	0	0	0		
Mammals	–	–	–	–	–	–	Report contains general discussion without specifying compounds.	60

Table 2
(Continued)

ENVIRONMENTAL DATA FOR SELECTED PAH

	Anthracene	Benz(a)-anthracene	Benzo(a)-pyrene	Chrysene	3-Methyl-cholanthrene	Phenanthrene	Other PAH, Comments	Report Page Reference
Occurrence/Variation								
Air	1	3	3	3	1	1	All PAH are found in all biomes and regions with some seasonal and geographical variation owing to variation in space heating and industrial distribution. Fossil fuel processing is single most important anthropogenic source and site of occurrence.	22,70,72
Water	2	3	3	3	2	2		23,74-76
Soil	1	3	3	3	1	1		23,77
Season	1	3	3	3	1	1		70-71
Geography	2	3	3	3	2	2		70,72
Plants/Food	2	4	4	4	2	2		78,80-84
Animals/Food	2	3	3	3	2	2		83
Fuels	2	3	3	3	2	2		85
Toxicity								
Bacteria	2	2	3	2	2	2	See p. 45-46 for list of other PAH cpds.	45,46
Algae	0	3	3	0	0	0	Phenanthraquinone, fluoranthene, 1,12-benzoperylene, 3,4-benzofluoranthene, indeno(1,2,3,cd)pyrene, 1,2,5,6-dibenzanthracene; (4)[a]	43,44
Higher Plants	2	0	2	2	0	2	Acridine, fluoranthene, 9-methylanthracene, 9,10-dihydroanthracene, 2-methylanthracene, 1,12-benzoperylene, 3,4-benzofluoranthene, indeno(1,2,3,cd)pyrene, 1,2,5,6-dibenzanthracene; (2)[a]	44
Invertebrates	0	0	4	0	3	0	Dimethylbenzanthracene, 2,7-diaminofluorene, N-fluoren-2-yl-acetamide; (2)[a]	46,47
Fish	3	0	3	3	3	3	Phenanthroquinone, with a rating of 3.	47,48
Amphibians	0	0	4	0	3	0	Dibenz(ah)anthracene, 1,2,5,6-dibenzanthracene; (3)[a]	48,49
Reptiles	0	0	0	0	0	0		
Birds	0	0	3	0	2	0		50
Mammals	3	3	4	0	3	0	9,10-dimethyl-1,2-benzanthracene, 1,2-benzopyrene. Primarily biochemical effects. (3)[a]	50,51,52

Table 2
(Continued)

ENVIRONMENTAL DATA FOR SELECTED PAH

	Anthracene	Benz(a)-anthracene	Benzo(a)-pyrene	Chrysene	3-Methyl-cholanthrene	Phenanthrene	Other PAH, Comments	Report Page Reference
Biosynthesis[d]								
Bacteria	–	–	–	–	–	–	Specific PAH compounds not mentioned in report. However, there is evidence that bacteria can synthesize PAH compounds.	62
Algae	0	0	0	0	0	0		
Higher Plants	2	2	3	0	0	0	Fluoranthene, 3,4-benzfluoranthene, indino(1,2,3,cd)pyrene, 1,2-benzperylene, pyrene, coronene, perylene, benzo(e)-pyrene. (2)[a]	61
Invertebrates	0	0	0	0	0	0		
Fish	0	0	0	0	0	0		
Amphibians	0	0	0	0	0	0		
Reptiles	0	0	0	0	0	0		
Birds	0	0	0	0	0	0		
Mammals	0	0	0	0	0	0		
Behavior in Biological Systems								
Adsorption/Distribution	2	2	3	2	2	2		63
Binding	1	3	4	1	2	1		64
Physico-Chemical Activity	2	2	2	2	2	2	Primarily general relationships.	65
Metabolic Activity	2	4	4	2	3	3	Data on alkyl PAH.	66
Structure-Activity	2	4	3	3	3	2	Data are available on several alkylated PAH including these six PAH.	67-69

[a] Code: 0 = no information, no inference possible; 1 = inference possible; 2 = minimal information; 3 = reliable data; 4 = reliable and extensive data.

[b] Report pages shown; where underlined indicates quantitative data.

[c] No data found for plants or animals.

[d] No data found for animals.

Regardless of natural PAH background levels and production mechanisms, it seems clear that industrial activity has increased the level present in the environment. PAH production is associated with automobile traffic (probably due to gasoline combustion, wearing of tires, and abrasion of asphalt surfaces), petrochemical manufacture, and fossil fuel combustion. PAH levels in urban situations are 10 to 100 times the levels found in remote areas. PAH levels in urban water supplies often exceed the level considered safe for human consumption.

Transport of PAH from water to air may be important in well-mixed water systems through distillation. In water and soil PAH occur almost completely as the absorbed state on minerals or organic particulate. In the air some PAH may be found in the vapor phase although most must be absorbed on particulate matter. In polluted rivers it is possible that PAH may be solubilized by micelles made up of lipids, biopeptides and alkaloids, but the relative importance of this mechanism in the total transport of PAH is unknown.

Chemical degradation of PAH in the environment can take place through a variety of oxidation reactions to give quinones as major primary products, with lesser amounts of diols, peroxides, and ring cleavage products. Some of these degradation products are more resistant to degradation than are the parent PAH, but their carcinogenic activity is generally much less than that of the parent PAH.

Some data are now available from which quantitative or semi-quantitative estimates may be made of half-lives of selected PAH in specific oxidation reactions. The major effect of structure on reactivity is increased reactivity of alkyl PAH toward electrophilic agents such as $RO_2\cdot$ radical, singlet oxygen, and ozone. Where data are available they point to reactivity factors (differences) as large as one hundred for alkyl PAH compared with the parent PAH. Chemical reactivity of PAH toward electrophilic agents seems to increase also with increasing numbers

of rings, but too few data are available on which to base any firm conclusions in this report.

Estimates of half-lives of selected PAH in reactions with $RO_2\cdot$ radical, singlet oxygen, ozone, chlorine, and HO· radical, the major environmental oxidizers, are summarized in Table 3. These data point to photooxygenation by singlet oxygen as being the dominant chemical process for degradation in water and probably also in air where reactions with HO· radical and ozone are also rapid. In the presence of both ozone and light, half-lives of a few minutes to a few hours may be expected for most PAH. In subsurface soil, microbial degradation is the major pathway for degradation and under anaerobic conditions no degradation of PAH occurs. In combustion processes where most PAH are probably formed, some degradation can also occur by reactions with nitrogen oxides and sulfur dioxide near the combustion zone.

Only a few studies have been conducted to assess the biological effects of polycyclic aromatic hydrocarbons other than those that relate to carcinogenicity, mutagenicity, or teratogenicity. These studies, however, indicate that these compounds can be acutely toxic to a variety of organisms throughout the phylogenetic scale and can produce a variety of sublethal effects. These effects, however, do not appear related in terms of degree or type to the number of rings, number of ring substituents, or their position, or the arrangement of the rings within the molecule.

Uptake of polycyclic aromatic hydrocarbons has been demonstrated in many types of microorganisms, plants, invertebrates, and vertebrates. In general, microorganisms, plants, and invertebrates tend to accumulate PAH compounds to a greater degree than vertebrates. The degree of accumulation appears to be related to the ability of the organism to metabolize PAH compounds; however, the relationship is clouded by evidence that some organisms may synthesize certain PAH compounds.

Table 3

RELATIVE RATES AND HALF-LIVES FOR DEGRADATION OF PAH BY ENVIRONMENTAL OXIDIZERS [a]

PAH	$RO_2\cdot$ [b]	Singlet Oxygen [c]	O_3, (Water) [d]	O_3, (Air) [c]	Cl_2 [f]	HO· [g]
Anthracene	1, 38,000	1, 5				
Dimethylanthracene		100, .05				
Diphenylanthracene		8, 0.6			All	
Phenanthrene	$2\cdot10^{-4}$, $2\cdot10^{8}$				PAH	
Pyrene	0.12, $2.4\cdot10^{5}$		1.5, 0.68	1.5, 560	have	
Perylene	1, 38,000				$t_{1/2}$ <0.5 hr	All PAH
Tetracene	400, 96	>1 <5				have
Benzopyrene	0.12, $2.4\cdot10^{5}$	1, 5	1, 1.05	1, 870		$t_{1/2}$~10 hr
Benzanthracene		2, 10	2.4, 0.45	2.4, 370		
Dimethylbenzanthracene		>1, <5				
Dibenzanthracene			2.5, 0.42	2.5, 340		
Dimethyldibenzanthracene		>1, <5	>6.2, 0.17	>6.2, <140		
Rubrene		300, .02				

[a] First figure in column is rate relative to standard rate of 1; second figure is half-life in hours.

[b] Table 8; same reactivities apply to air; [c] Table 9; same as b; [d] Table 12; $[O_3] = 10^{-4}$ M; [e] Table 13; $[O_3] = 2\cdot10^{-9}$ M

[f] For 10^{-5} M Cl_2, benzpyrene most reactive; [g] Assumes $k = 10^{9}$ 1 mol^{-1} sec^{-1}.

This often makes it difficult to determine whether the levels found in organisms are of exogenous or endogenous origin.

Aquatic algae and invertebrates tend to concentrate PAH compounds. In areas where measurable amounts of these compounds have been found in the water, the concentration of the compounds in algae and invertebrates has been found to exceed that of the water by at least 200 times. Concentrations in terrestrial plants usually parallel that in the soil; however, plant levels are usually lower than soil levels.

No relationship appears to exist between the molecular structure or the number of benzene rings of PAH compounds and their propensity for accumulation. A similar lack of relationship is found for structures of PAH and their partition coefficients (Tables 4 and 5). However, the data in Table 4 have been interpreted to mean that those PAH with the highest carcinogenicity also exhibit the highest solubility in aqueous soaps.

Biodegradation of polycyclic aromatic hydrocarbons has been demonstrated in microorganisms, fish, birds, and mammals. The aryl hydrocarbon hydroxylase enzyme system concerned in the metabolism of PAH compounds seems to be the same in all organisms. Although metabolism of these compounds by plants and invertebrate animals has not been demonstrated, it is likely that metabolism does occur.

The rate of degradation of PAH compounds by microorganisms to mammalian systems is relatively low. Bacteria found in soil or water containing PAH compounds tend to metabolize these compounds at a much faster rate than those that come from relatively noncontaminated areas.

Unlike chemical degradation, susceptibility of PAH compounds to biodegradation does not appear related to structure or number of rings. Non-adapted sewage sludge microorganisms readily attack phenanthrene, but metabolize anthracene, another tricyclic compound, to a limited extent. Of the tetracyclic compounds, 9,10-dimethylbenzanthracene is much more susceptible to oxidation than 1,2-benzanthracene and 9,10-dimethyl-1,2-benzanthracene.

Table 4

PARTITION COEFFICIENTS OF POLYCYCLIC HYDROCARBONS IN A HEXANE-MONOETHANOLAMMONIUM DEOXYCHOLATE SYSTEM AND THE APPROXIMATE CARCINOGENIC ACTIVITIES[a]

Compound	Partition coefficient	Approximate carcinogenic activity
20-Methylcholanthrene	5.8	++++
Benzoperylene	5.7	–
1,2,4,5-Dibenzopyrene	6.8	+++
1,2,5,6-Dibenzanthracene	6.9	+++
3,4-Benzopyrene	7.9	++++
2,3-Benzofluorene	8.4	–
10-Methyl-1,2-benzanthracene	8.7	++++
1-Methylphenanthrene	10.1	–
1,2-Benzanthracene	10.1	+
10,11-Benzofluoranthene	10.2	++
Chrysene	10.4	+?
1-Methyl-3,4-benzophenanthrene	10.9	++
Pyrene	11.5	0
Fluoranthene	12.2	0
1-Methylpyrene	12.3	–
Anthanthrene	12.5	0
Retene	12.7	0
2-Methylanthracene	13.6	0
7-Methyl-3,4-benzophenanthrene	14.4	+
3'-Methyl-1,2-benzanthracene	14.5	0
3-Methylpyrene	14.6	–
Naphthacene	14.7	0
4-Methylpyrene	14.9	0
Phenanthrene	16.4	0
Anthracene	16.9	0
2-Methylfluorene	17.9	–
Fluorene	18.7	0
Naphthalene	24.4	0

[a] In part from R.R. Demisch and G.F. Wright, Can. J. Biochem. Physiol. 41, 1655(1963). "–" designates compounds not tested for carcinogenicity. The partition coefficient represents the ratio of concentration in the hexane phase over that in the deoxycholate phase, determined spectrophotometrically.

Source: Chemical Induction of Cancer, Vol. IIA, J.C. Arcos and M.F. Argus, Academic Press, N.Y. 1974.

Table 5

PHYSICAL PROPERTIES OF SIX PAH

PAH	Mol. Formula	Mol. Wt.	M.P. °C	B.P. °C/torr	Log Partition Coefficient[a]
Benzo(a)pyrene	$C_{20}H_{12}$	252.30	179 [b]	311/10 [b]	6.04 [c]
Benzo(a)anthracene	$C_{18}H_{12}$	228.28	160 [b]	400/760 [b]	5.61 [c]
Methylcholanthrene	$C_{21}H_{16}$	268.34	179 [d]	NA	5.83 [c]
Chrysene	$C_{18}H_{12}$	228.28	256 [b]	448/760 [b]	5.61 [c]
Anthracene	$C_{14}H_{10}$	178.23	216 [e]	340/760 [e]	4.45 [f]
Phenanthrene	$C_{14}H_{10}$	178.23	101 [e]	340/760 [e]	4.46 [f]

[a] Partition Coefficient = $[PAH]_{1\text{-Octanol}} / [PAH]_{H_2O}$

[b] IARC 1973

[c] Hansch 1975

[d] Fieser 1935

[e] Handbook of Chemistry & Physics 1964

[f] Leo 1971

In mammals and birds, studies on degradation have been limited to 3,4-benzopyrene and 3-methylcholanthrene; hence, the relationship between compound structure and metabolism in these animals cannot be evaluated.

Microorganisms are capable of completely assimilating PAH compounds and appear to utilize them as a carbon source. Mammals, on the other hand, oxidize these compounds to the epoxides or hydroxylate them and eliminate these metabolites via the urine and feces. Some epoxides and hydroxylated derivatives of PAH compounds are carcinogenic or mutagenic, but less so than the parent compounds. Although the hydroxylated derivatives have been isolated from bacteria, it is not known whether bacteria form epoxides. The biological activity of metabolites other than the epoxides and hydroxylated compounds is not well defined.

Our review of the literature on PAH compounds in terms of toxicity, bioaccumulation, and biodegradation has revealed that relatively little is known about these compounds--particularly in lower forms of life. Few experimental investigations have been performed.

Among unsubstituted 3-5 ring PAH, physical properties, absorption, distribution, binding to protein and nucleic acid and metabolic transformations are relatively similar. Differences in diverse biological effects relate frequently to differences in carcinogenicity. Table 4 gives some partition coefficients and carcinogenic activities for selected PAH. All of the compounds are readily absorbed by biological systems due to their high lipid solubility and aqueous solubilization by lipids and macromolecules. High lipid solubility insures efficient microsomal metabolism, which produces both reactive, cytotoxic intermediates and inactive polar metabolites, which are rapidly eliminated. High lipid solubility also determines efficient uptake and prolonged storage in fat deposits, thus providing a reservoir for continuous release and metabolism over a prolonged period. This results in prolonged, constant exposure to multiple PAH with enzyme induction and competitive interactions, which may be protective or synergistic with regard to chronic toxicity.

Structure-activity relationships remain poorly defined but are related primarily to geometry, positional electron density, and reactivity, which determine metabolic transformations that result in reactive intermediates that are either rapidly metabolized further and eliminated or function as tissue alkylating and intercalating cytotoxins. Some, but not all, alkyl derivatives of PAH are more potent carcinogens and mutagens than the unsubstituted hydrocarbons. The preponderance of studies center on benzanthracene, benzopyrene and, to a lesser extent, methylcholanthrene.

Recommendations

Direct analyses for vapor concentrations of PAH and for the proportion of PAH in the vapor phase and on particulates are needed. Measurements of this kind should be carried out along combustion plumes as a function of distance from the source, particle size distribution, and plume temperature.

Distillation of PAH from water to air should be evaluated for several PAH, as should transfer mechanisms to and from water and soil.

Most chemical studies have examined only a few PAH by themselves under laboratory conditions; much more emphasis should be placed on studies of the rates of degradation of selected mixtures of PAH under environmentally useful conditions.

Structure-reactivity studies on PAH are needed, with particular emphasis on effects of alkyl substituents and ring members on reactivity under conditions where the useful kinetic data can be obtained. Cooxidations of these PAH in mixtures should be part of such studies.

Although PAH compounds are taken up and accumulated by many organisms, we do not know if consumer organisms, particularly the primary consumers, take up these compounds directly from the environment or through the food chain.

Metabolism of PAH compounds has been established in microorganisms and the higher vertebrates, but so far, not in invertebrates. Comparative metabolism studies could be helpful in understanding the mechanism of PAH biodegradation as well as the environmental fate of these compounds.

There appears to be little information on the acute toxicity of PAH compounds to aquatic organisms. Although most of these compounds have limited water solubility, it has been demonstrated that as little as 40 μg/l of phenanthraquinone can be toxic to algae. Only a few compounds have been tested for toxicity to fish, and none have been tested for toxicity to aquatic invertebrates.

III LITERATURE SEARCH

Sources and Subject Area

Chemical Abstracts was searched from 1965 through May 1975 for biological and chemical activity of the six compounds under study. Part of this was by manual search and the last 4-5 years (1971-1975) was carried out using the Systems Development Corporation (SDC) computerized files of Chemical Abstracts. The U.S. Government Reports file was searched by using DIALOG computerized source. Other sources, such as Biological Abstracts, Current Contents, and Selected Water Resources Abstracts were searched manually.

Searching was done (1) by the Chemical Abstract Service number for each compound, (2) on synonyms for each compound, and (3) by such terms as environmental fate, biodegradation, toxicity, polynuclear/polycyclic aromatic hydrocarbons, and coal tars as well as by the correct chemical name.

In addition to the abstracts searched, references in pertinent articles were scanned for further information, and calls to a selected number of scientists working in this area were made to elicit additional data or references.

Results

The number of references culled in the first search was overwhelming. In the computerized search alone, approximately 3000 citations were retrieved. Manual searching of other sources added about 600-700 more references. Of these, approximately 1000 were selected for further study by the panel of experts. Full-text copies of articles that appeared to be of interest were ordered. Approximately 300 articles were ordered.

IV REVIEW AND EVALUATION OF LITERATURE

Formation and Degradation of PAH Under Environmental Conditions

A comprehensive review of the occurrence and properties of many PAH was prepared by the National Academy of Sciences (NAS) in 1972. Although quite complete in many respects, that review examined only qualitatively the chemical and physical properties of PAH as related to their environmental fate and lifetime. This review attempts to provide a more quantitative framework for environmental fate and lifetime based on our current knowledge of the dominant chemical reactions in air and water responsible for removal of PAH, specific rate constants for these reactions and the probable concentrations of reactive intermediates. It is important to note that there are very few published reports of lifetime experiments under environmental conditions and the estimates reported here are calculated from composite sources. As such these values for lifetime are probably accurate to within an order of magnitude (sometimes better). Nonetheless, competing processes are often so slow that in many cases these values can be used to provide a reliable model of environmental degradation.

Physical Properties and Transport

The physical properties of many PAH, which largely determine their rates and mode of transport between air, water, and soil, are fairly well characterized except for specific absorption properties. Tables 5 and 6 summarize some of these important properties.

All of the PAH are high melting/high boiling solids with very large values for partition coefficients indicative of significant accumulation and concentration in biological (lipid) material. These partition values may be misleading, however, since very little if any PAH are actually found dissolved in water. Recent observations of Andelman (1971) and McGinnes (1974 a,b) suggest that PAH occur either as very finely dispersed particles in water or adsorbed on a variety of particulates such as

Table 6

VAPOR PRESSURE AND VAPOR CONCENTRATION OF SELECTED PAH AT 25°C

	Vapor Pressure Torr[a]	Vapor Concentration[b]	
		$\mu g/10^3\ m^3$[b]	moles/ℓ
Benzo[a]pyrene	5.49×10^{-9}	75	2.97×10^{-14}
Benzo[a]anthracene	1.10×10^{-7}	1333	5.84×10^{-14}
Benzo[e]pyrene	5.54×10^{-9}	75	2.97×10^{-14}
Benzo[k]fluoranthrene	9.59×10^{-11}	13	5.15×10^{-15}
Benzo[ghi]perylene	1.01×10^{-10}	1.5	5.43×10^{-16}
Coronene	1.47×10^{-12}	0.02	6.66×10^{-18}
Anthracene	1.95×10^{-4}	1.87×10^{7}	1.05×10^{-8}
Phenanthrene	6.80×10^{-4}	6.51×10^{7}	3.65×10^{-8}
Pyrene	6.85×10^{-7}	74,400	3.68×10^{-11}

[a] All data from Pupp 1974 except for anthracene and phenanthrene, Jordan (1954). The equations given in the references were determined from data obtained at 100-300°C or higher; vapor pressures at 25°C are extrapolations.

[b] Calculated from data in Pupp 1974 except for anthracene and phenanthrene which were calculated from equations in Jordan 1954 and the ideal gas law.

minerals or carbonized materials. Solubilities in water of most PAH with more than three rings appear to be too small to measure, that is, less than 10^{-10} M.

Solubilization of PAH in water by micellar mechanisms involving surface active species such as detergents, biopeptides, and alkaloids, are suggested by Eisenbrand (1971) and Demisch (1963) as possible modes of transport for PAH in natural waters. Available data do not provide convincing evidence that these laboratory-observed solubilization mechanisms are very important in natural waters where solubilizer concentrations are likely to be much lower than particulate concentrations. The extremely large amounts of PAH, such as benzo(a)pyrene, found in river bottoms suggest that adsorption on particulates and sediment clays is a major process for removing these materials from water systems.

Recent data on vapor concentrations of PAH are of particular interest in connection with transport and analysis of PAH in the air. Table 6 summarizes the data of Murray, Pottie, and Pupp (1974) and of Pupp et al (1974). Vapor pressures for PAH at 25°C are extremely low, ranging from $6.8 \cdot 10^{-4}$ torr for phenanthrene to $1.5 \cdot 10^{-12}$ torr for coronene. Nevertheless, the equilibrium vapor concentrations of some of the more volatile PAH such as anthracene, phenanthrene, pyrene, and benzanthracene are sufficiently high to lead to significant errors in analyses for air-borne PAH where analyses are based only on collected particulates. The data indicate that for benzopyrene, benzanthracene, and benzofluoranthrene the equilibrium vapor concentrations can easily exceed the values reported for these PAH in air, based on particulate sampling (Hoffmann, D. 1968). However, in cases where PAH are quickly adsorbed by air-borne particulates just beyond the combustion zone, then the actual concentrations in the vapor state are likely to be much lower, thereby reducing the analytical error.

Recently Mackay and Wolkoff (1973) have developed a quantitative treatment of the rate of transport of low-volatile organics from water to air. Their results show that some high molecular weight organics such as Aroclor 1260 (mw 361; vapor pressure $\sim 4 \cdot 10^{-5}$ torr; solubility $\sim 7 \cdot 10^{-9}$ M) have surprisingly low half-lives in water (28 min for Aroclor) owing to exceptionally high activity coefficients. No calculations of this kind have been applied to PAH; it is likely that adsorption of PAH on surfaces will significantly reduce the activity coefficients. which will slow the rate of codistillation. However, some measurements of rates of distillation of PAH seem warranted.

Spectra

Spectral properties of six PAH summarized in Table 7 show that all but one of these compounds absorb solar radiation strongly above the solar cutoff (300 mm), and in some cases well into the visible region. This is one of the most important properties of PAH since it provides the basis for efficient self-initiated photooxidation, a process discussed in more detail below.

Dissimilarities in uv spectra have provided the basis for sensitive analysis of PAH, particularly since log ϵ is so large (Table 7). Fluorescence spectra of many PAH are also well characterized and provide the basis for detection at concentrations of 0.01 - 0.001 the values useful for adsorption spectra (NAS, 1972). However, it is clear that for a natural system in which as many as several hundred PAH homologs, analogs, and isomers may be found in a single sample, spectroscopic techniques alone are not incisive enough to distinguish one PAH from another, and additional separation and identification by GC/MS is now routinely used along with fluorescence.

Table 7

SPECTRAL PROPERTIES OF SIX PAH

	λmax_{nm}	log ϵ	Solvent	Ref.
Benzo(a)pyrene	347	4.12	EtOH	a, p. 839
	364	4.38		
	384	4.48		
Benzo(a)anthracene	314	3.67		
	327	3.81	EtOH	a, p. 751
	341	3.87		
	359	3.72		
	376	2.73		
	386	3.86		
Methylcholanthrene	no absorption above 300 nm			a, p. 892
Chrysene	344	2.88	EtOH	b, p. 251
	351	2.62		
	360	3.00		
Anthracene	308	3.15		
	323	3.47	EtOH-	b, p. 291
	338	3.75	MeOH	
	355	3.86		
	375	3.87		
Phenanthrene	309	2.40		
	314	2.48	EtOH-	b, p. 228
	323	2.54	MeOH	
	330	2.52		
	337	3.40		
	345	3.46		

Refs: a. Organic Electronic Spectral Data, Vol. I. Interscience Publishers, Inc., New York, 1960, Mortimer J. Kamlet, Ed.

b. Polycyclic Hydrocarbons, E. Clar. Academic Press, London, 1964.

Formation of PAH

The ubiquity of PAH in the environment is well documented; Table 8 summarizes several studies on occurrence of PAH in the air, water, and soil, and the review by Andelman and Suess (1970) provides a very thorough review of PAH in water. Although anthropogenic combustion sources are most commonly cited as the major source of PAH, natural combustion sources (Blumer 1975) and microorganisms may also play significant roles in their generation. Certainly conditions found in forest fires are compatible with requirements found for conversions of simple aromatics and hydroaromatics to PAH under laboratory conditions, namely temperatures between 500 and 900 ^{o}C and pyrolyzing rather than oxidizing environments. Pyrolysis of lignins and terpenes during forest fires is a likely source of PAH in relatively high yields (Dikun 1965, Levin 1965, Liverovskii 1972). Foodstuffs, including lipids and cholesterol (Halaby 1971) and amino acids and carbohydrates (Masuda 1967), are also probable sources of PAH when pyrolyzed at 300 - 700 ^{o}C.

The chemical mechanisms by which PAH are formed by pyrolysis of simpler organic structures are extremely complex. Badger's (1964a,b,c, 1966) extensive studies on pyrolysis of aliphatic, aromatic, and olefinic compounds to PAH indicate that two- and three-ring structures arise from cyclization of side chain radicals with aromatic rings, and that polycyclic aromatics form by dimerization of di- and tricyclic aromatic or hydroaromatic rings.

Blumer (1975) has recently suggested that PAH from natural combustion sources, i.e., forest fires, are structurally distinct from PAH produced from anthropogenic sources in having a high proportion of alkyl side chains owing in part to the lower temperatures found in natural fires. This is an interesting suggestion, which, if correct, could provide a

Table 8

CONCENTRATIONS OF PAH IN AIR, WATER, SOIL

PAH	Air[a] $\mu g/10^3 m^3$	Surface Water[b] $\mu g/l$	Soil[c] $\mu g/kg$
Benzo[a]pyrene	0.2 - 39	0.002	75-370[d]
Benzo[a]anthracene	0.1 - 22	0.014	41-330
Methylcholanthrene	[e]	[e]	[e]
Chrysene		0.038	40-240
Anthracene			8-170
Phenanthrene			33
Benzo[e]pyrene	1 - 26		59-310
Benzo[k]fluoranthrene	1 - 20		
Benzo[ghi]perylene	2 - 46		66-280
Coronene	0 - 20		5-20
Pyrene	1.3 - 35	0.110	100-960
Fluoranthene		0.128	110-790
Perylene			26-94

[a] D. Hoffmann 1968

[b] Borneff 1964

[c] Giger 1974

[d] Values as high as 15000 mg/kg reported in some sediments, Andelman 1970

[e] Occurrence mentioned in several references, but no quantitative data were found. See M. Kertesz-Saringer 1972b (air); L. Zoccolillo 1972 (airborne particulates); and M.I. Stepanova 1972 (waste water).

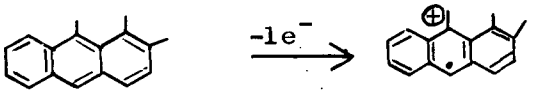
valuable means of distinguishing the natural burden of PAH (background) in the environment from that imposed by man, both on a regional and continental basis.

However, no published data are available that either corroborate or refute Blumer's idea, and many of the older data (pre-1972) are based on analytical techniques that fail to distinguish between alkylated and non-alkylated PAH. Sawicki (1975) has expressed some reservations about the results, based on the complexity of the analytical schemes employed by Blumer.

Blumer's argument that natural combustion sources produce more alkylated PAH because of lower temperatures where alkylated PAH are possibly more stable may be misleading, since the rates of reaction of PAH are governed by both temperature and time. Products from natural combustion processes probably have longer residence times in the pre-flame region than do products from utility boilers or automotive engines, possibly enough longer to compensate kinetically for lower temperatures. Since nothing is really known about how alkyl PAH are formed or their rates of reaction in combustion systems (they may or may not oxidize much more rapidly), the validity of Blumer's idea must be established by extensive and careful analyses of PAH from a variety of natural and anthropogenic sources.

Chemical Degradation of PAH

PAH are highly reactive compounds that undergo all the reactions commonly found for simple aromatics as well as a number of reactions that result from facile removal of one electron from the polycyclic system to form a radical cation.



Further reaction of the radical leads to oxidized products, including endoperoxides, diols, quinones, and dimers. As expected, electron-donating groups (alkyl, alkoxy) accelerate the rates of these reactions. The NAS report (1972) provides a good review of the qualitative features of these reactions, as does an earlier report by Tipson (1965), which reviews oxidation reactions of PAH, particularly those using metal ion oxidizers.

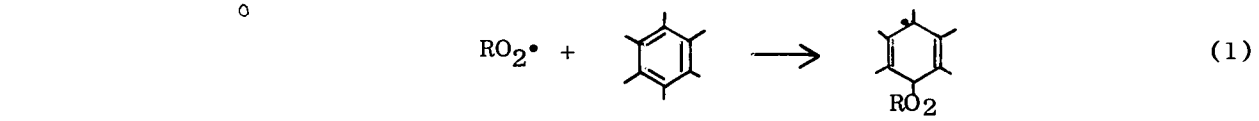
Rates of and Mechanisms of Degradation in Water

In some respects, reactions of PAH in natural waters are somewhat simpler than in air owing to the presence of fewer kinds of oxidizing species. In natural water the principal oxidizing species are (1) alkylperoxy and hydroperoxy radicals ($RO_2\cdot$, $HO_2\cdot$), generated by photolytic cleavage of trace carbonyl compounds or from enzymatic sources. [Radical reactions involving oxygen are termed autoxidations.] (2) singlet oxygen generated in a variety of reactions involving oxygen (ground-state) with excited singlet and triplet species that are formed mostly (but not exclusively) by light absorption by PAH. [Singlet oxygen ($^1O_2$) reactions are generally termed oxygenations or, where light is used, photooxygenations.] Some quantitative rate data are available for estimating half-lives of reactions of PAH with $RO_2\cdot$ radicals and singlet oxygen. Thus, the relative contributions of the two processes to degradation of PAH may be compared.

Since most urban drinking water is treated with chlorine or ozone, it is also of interest to try to estimate their effects on PAH lifetime and compare them with autoxidation and photooxygenation.

Autoxidation--several qualitative studies of autoxidation of PAH have been reported (NAS, 1972; Tipson, 1965). Although in most cases detailed product analyses are not available, the substitution pattern

for reaction of $RO_2\cdot$ radicals on the PAH ring should be much the same as for other electrophilic species including $^1O_2$, NO_2^+, and O_2. Mahoney (1964, 1965, 1975) has measured the rates of autoxidation of several PAH under conditions where the rate constants for reaction of $RO_2\cdot$ and PAH can be evaluated.



(1)

Table 9 summarizes specific rate constants for reaction (1) and half-lives for several PAH at 60°C. Half-life values for oxidation are based on reaction (1) and the rate law

$$-d\ PAH/dt = k_p[RO_2\cdot][PAH]$$

Thus, the half-lives depend only on values for k_p and $[RO_2\cdot]$. We have assumed that $[RO_2\cdot]$ has a steady-state value of 10^{-10} M, under average daily illumination in natural water systems.*

$$t_{1/2}/days = 0.69/k_p\ 10^{-10}$$

The surprising result is that anthracene is so much more reactive than phenanthrene and so much less reactive than tetracene and that the other tetra- and pentacyclics are so unreactive compared with tetracene. Differences in rates of reaction of PAH with $RO_2\cdot$ are probably as large as found for any group of organic compounds. For all PAH in Table 9, except for tetracene, half-lives for radical oxidations are so long even at 60°C that other, faster processes must intervene to remove them from the environment. Experiments are needed to measure the value of

*This estimate is based on assumptions concerning average carbonyl concentrations, quantum yields for radical formation and photon fluxes in the absorption bands of interest.

Table 9

ABSOLUTE RADICAL CONSTANTS AND HALF-LIVES
FOR REACTION OF $RO_2\cdot$ RADICAL WITH PAH AT 60°C

PAH	k_p/l mol^{-1} sec^{-1}	$t_{1/2}$/day [a]
Anthracene	50 [a,b]	1600
Phenanthrene	<0.01 [c]	>8.10^6
Tetracene	20,000 [b]	4
Benzo(a)pyrene	6 [c]	9900
Perylene	50 [a]	1600
Pyrene	6 [a]	

[a] Mahoney 1965

[b] Mahoney 1964

[c] Mahoney 1975

k_p for other PAH and to estimate more reliably the range of concentrations of $RO_2\cdot$ in natural water systems.

Photooxygenation--The term photooxidation is commonly used to describe all reactions with oxygen that require light, regardless of the reactive species involved. The bulk of the evidence suggests that most photooxidations of PAH involve reaction with singlet oxygen (photooxygenation); however, generation of and reaction with peroxy radicals is also possible via radical-cation intermediates (NAS, 1972).

Singlet oxygen can be generated by a variety of chemical and light-sensitizer reactions. Gollnick and Schenck (1968) have reviewed early work on the reactions of PAH with singlet oxygen, and the NAS report summarizes the results. Products of photooxygenation vary with the structure of the PAH; in cases where 9,10 positions are open (as in anthracene), an endoperoxide is the primary product; but in some cases it is unstable, and a quinone is the first isolable product. Benzo(a)-pyrene photooxygenates to the same mixture of diones formed by oxidation with one-electron oxidizers such as Cr(VI) (Antonello 1964). Although singlet oxygen seems the likely oxidizer, the intermediate is probably not an endoperoxide.

Of particular interest is (1) how the reactivities of different PAH toward singlet oxygen are affected by structure and (2) how rapidly PAH photodegrade under environmental conditions.

The detailed kinetic scheme for self-sensitized photooxygenation of a PAH can be generalized following Stevens and Algar (1968)

$$^1M^* \rightarrow M + h\nu \qquad (1)$$

$$^1M^* \rightarrow {}^3M^* \qquad (2)$$

$$^1M^* \rightarrow M \qquad (3)$$

$$^1M^* + {}^3O_2 \rightarrow {}^3M^* + {}^3O_2 \qquad (4)$$

$$^1M^* + {}^3O_2 \rightarrow {}^3M^* + {}^1O_2 \qquad (5)$$

$$^3M^* \rightarrow M \qquad (6)$$

$$^3M^* + {}^3O_2 \rightarrow M + {}^1O_2 \qquad (7)$$

$$^3M^* + {}^3O_2 \rightarrow M + {}^3O_2 \qquad (8)$$

$$M + {}^1O_2 \rightarrow MO_2 \qquad (9)$$

$$^1O_2 \rightarrow {}^3O_2 \qquad (10)$$

where the asterisk denotes electronic excitation, M, $^1M^*$, $^3M^*$ are PAH and $^3O_2$ and $^1O_2$ are triplet (ground-state) and singlet oxygen, respectively.

Oxygen plays several roles in the overall scheme, including energy transfer and collision-induced intersystem crossings of $^1M^*$ to $^3M^*$ (steps 4 and 5) as well as reaction with $^3M^*$ to form products (step 9). The ratio k_{10}/k_9, often referred to as β, has been evaluated independently from other steps in the process (Foote 1972). Thus values for k_9 can be estimated as shown in Table 10.

Table 10 summarizes some relative and absolute values for k_9 for several PAH. Data are sparse, and in some cases only qualitative comparisons are possible. The relation between structure and reactivity of PAH is not clearly defined by these data. However, the effect of the methyl groups clearly seems to accelerate the reaction.

Table 10

RELATIVE AND ABSOLUTE REACTIVITY OF PAH
TOWARD SINGLET OXYGEN

PAH	Rel. Reactivity[a] k/k_o	Abs. Reactivity[b] k_2/1 mol^{-1} sec^{-1}	Ref.
Anthracene	1	2.10^6	Foote 1972
Dimethylanthracene	100	2.10^8	Corey 1964
Diphenylanthracene	8	1.710^7	Bowen 1954
Rubrene	300	$6 \cdot 10^8$	Bowen 1954
Benzo(a)pyrene	~1	$\sim 2 \cdot 10^6$	Kuratsune 1962
Naphthacene	>1	$>2 \cdot 10^6$	Kuratsune 1962
Dimethyldibenzanthracene	>1	$>2 \cdot 10^6$	Bowen 1954

[a] Estimated roughly against anthracene.

[b] From β values given in references and $k_{10} = 10^5$ sec.

The actual rate of reaction (9) depends on the concentrations of PAH and singlet oxygen, and the concentration of singlet oxygen will depend on many factors including light flux, the quantum yields for conversion of one excited species to another (including formation of singlet oxygen in steps 5 and 7), and the rates of efficiencies of competing deactivation processes such as steps 3, 6 and 7. Algar and Stevens (1970) have examined several different mechanisms for inhibition of photooxygenation in solutions of rubrene, dimethylanthracene, and dimethyldibenzanthracene. In each case the role of oxygen in promoting and/or inhibiting the quantum yield for photooxygenation is different.

Since the PAH occur in the environment as complex mixtures almost always adsorbed on organic and mineral surfaces, the applicability of these studies to environmental conditions is of limited value other than to indicate some of the basic mechanistic features, the likely complications that may occur, and the danger of generalizing from one set of experimental data. It is therefore of some interest to examine two recent kinetic studies in which benzo(a)pyrene and benzo(a)anthracene were photolyzed in water, either dispersed as microspheres or adsorbed on mineral surfaces. Although neither study examined any mixtures of PAH, these data are closer to an environmental situation.

McGinnes and Snoeyink (1974) carried out a study using benzopyrene and benzanthracene dissolved in acetone:water (20:80), dispersed in water alone, and adsorbed on kaolin. Results summarized in Table 11 show that in acetone: water, the rates of oxidation of the two PAH are nearly linear functions of light intensity and, at about a third to a fourth the intensity of sunlight (1.0 - 1.3 mW/cm^2), the rates of oxidation of benzopyrene and benzanthracene are similar with first order rate constants k_1 = 0.29 and 0.44 hr^{-1}, respectively. The rate law is

Table 11

PHOTOOXYGENATION OF BENZO(A)PYRENE (BaP) AND BENZ(A)ANTHRACENE (BaA) IN WATER AT 25°C [a]

PAH	Particulate Form/Conc.	Light Flux mw/cm^2	k_1/hr	$t_{1/2}$ hr
BaP	Solution in	0.13	0.009	76
	20% acetone:	0.48	0.098	7.0
	water	1.15	0.29	2.4
	1 mg/l			
BaP	Microspheres	0.61	0.057	12
	1.5 μm dia	1.37	0.057	12 [c]
	1 mg/l [b]			
BaA	Solution in	0.13	0.00	
	20% acetone:	0.48	0.10	6.9
	water 1 mg/l	1.37	0.44	1.6
BaA	Microspheres	0.61	0.028	25
	1.5 μm dia	1.31	0.049	14
	1 mg/l [b]			

[a] McGinnes 1974

[b] Refers to amount in total solution.

[c] Reaction inhibited at 60% decomposition; second half-life was 3.5 days.

of the form

$$- d(PAH)/dt = kI[PAH]$$

where I is light flux.

Significant differences in rates were found for the two PAH when they were suspended in water as microspheres of ~1.5-mm diameter. Benzopyrene disappears very rapidly until, when nearly 60% decomposes in about 8 hours, the process nearly stops and only 20% more decomposes in the next 4 days at the highest light flux, 1.34mW/cm^2. Benzanthracene, on the other hand, shows a first order decomposition to nearly exhaustion of benzanthracene with a life-life of ~14 hours at 1.37 mW/cm^2 light flux and nearly linear dependence on light intensity.

Not surprisingly, some data show that increased surface area increases the rate of oxidation. When adsorbed on kaolin and suspended at different loadings in water, both benzopyrene and benzanthracene decomposed at similar rates, despite the fact that light scattering was 100-fold greater in the high loading samples.

Andelman and Suess (1971) carried out similar studies on benzopyrene dissolved in acetone and adsorbed on $CaCO_3$ at several temperatures and with variable oxygen concentrations. For the most part, their findings agree with those of McGinnes and Snoeyink (1974) and elaborate on them. One experiment using benzopyrene adsorbed on $CaCO_3$ at 0.31 mW/cm^2 light flux gave $t_{1/2}$~25 hours at 21°C.

The temperature dependence for this oxidation is summarized in Table 12. The apparent activation energy for the process is 15 kcal/mole, corresponding to a ten-fold change in rate going from 0° to 25°C.

Andelman (1971) found a small effect of oxygen concentration on the rate corresponding to $[O_2]^{0.1}$. This effect could well be due to a balance between inhibition and activation processes discussed earlier. The mechanistic significance was not elaborated.

Table 12

TEMPERATURE DEPENDENCE FOR PHOTOOXYGENATION OF BENZOPYRENE ON $CaCO_3$ [a,b]

t, °C	k_1/hr	$t_{1/2}$, hr
5	0.0019	360
21	0.0087	79
31	0.022	31

[a] Fieser 1935

[b] 5 g $CaCO_3$ with 1 μg/g benzpyrene was illuminated with a 1.3 mW/cm^2 flux for 7 hours.

Their data indicate that rates of photooxygenation of benzopyrene will decrease dramatically in winter months even under clear skies or when oxygen concentrations are very low, a conclusion that is consistent with other observations (see section on Environmental Sources). The reasons for the temperature and oxygen dependence need to be clarified so as to be able to predict more accurately how environmental conditions will affect rates for different PAH.

The autoinhibition found for benzopyrene microspheres but not for its solution nor for benzanthracene in either form seems to be an important observation in connection with environmental degradation of PAH. The mechanism of inhibition in the microspheres may be simply protective absorption of light by the product quinones, as suggested by McGinnes (1974), or it could be a more complex inhibition scheme involving energy transfer or quenching peculiar to the solid state. Other PAH may also exhibit this effect and more importantly, in mixtures of PAH that are found in nature, inhibition by benzopyrene quinones could inhibit the degradation of other PAH which would, by themselves, undergo more rapid and complete degradation. Clearly more experimental work is needed to answer some of these questions. The important conclusion that emerges from these two studies is that under environmental conditions, benzo(a)-pyrene and benz(a)anthracene undergo rapid photooxygenation when suspended or adsorbed on mineral surfaces.

A more recent study (Katz and Lane, 1975) on photodegradation of thin films of solid BaP under simulated smog conditions shows that high ozone levels (2 ppm) markedly reduce the halflife to a few minutes in full sun.

McGinnes (1974) examined briefly the products from his experiments and found the same quinones reported by others (NAS, 1972). In the case of benzanthracene, its primary products (mostly quinones) began to decompose when about 90% of the parent PAH had disappeared. The results suggest that the products are much less susceptible to photooxygenation

than are the parent PAH, and that in many situations, these products will remain for some time unless removed by some other alternative process. Here again, additional work is required to evaluate, in a cooxidizing system, the relative reactivities of different PAH and their products.

In addition to autoinhibition, another complication that must be borne in mind when trying to interpret qualitative photochemical environmental observations, with which the literature abounds, is that other kinds of compounds also commonly found in water, soil or air can affect the photooxygenation of PAH. For example, Gubergrits, Paalme, and Kirso (1972) claim that phenols in water changed the kinetics and mechanism of photodegradation of benzopyrene with the net effect, among several competing reactions, of inhibiting photodegradation.

Some metal-ion complexes are well known (Guillory 1973) quenchers for singlet oxygen; their presence as adventitious impurities along with PAH could reduce significantly the rate of photooxygenation of such mixtures. Although 3-methylcholanthrene does not absorb (Table 7) above the solar cutoff (300 nm), it may be photooxidized where $^1O_2$ is produced by other PAH present in natural mixtures.

Reactions with Chlorine and Ozone

Chlorination or ozonization of urban drinking water is used to kill pathogens, but the chlorine and ozone must also chemically interact with organics such as PAH. One report by Trakhtman and Manita (1966) indicates that one microgram of benzopyrene was reduced to 0.188 microgram in 30 minutes and to 0.06 microgram in 2 hours using $7 \cdot 10^{-16}$ M (0.5 mg/l) chlorine in water. These data do not fit any simple kinetic scheme but correspond to an initial ten-minute half-life, followed by a 30-minute half-life for the next increment. The results are supported by some semi-quantitative studies by Sforzolini et al (1973) who examined five PAH in water also containing chlorine at $7 \cdot 10^{-6}$ M. In 30 minutes all benzopyrene was consumed; lesser amounts of other PAH, but in all cases over 50%, were consumed in similar experiments.

Ozone is commonly used for water treatment in Europe and is applied in a batchwise manner at levels of 5 ppm. Products of reaction of ozone with PAH in solution have been examined in detail, and the results are summarized in the NAS report (1972).

The relative and absolute reactivities of PAH toward ozone do not appear to have been determined in any systematic or quantitative way. The best guess that can be made is based on the observation of Il'nitskii et al. (1968), who measured the amounts of PAH remaining after treatment of $0.67 \cdot 10^{-5}$ g/l of PAH with 0.4 g/l of ozone for one minute at 25°C. From these data we can calculate rough rate constants for reaction of 5 ppm ozone with PAH, which are summarized in Table 13.

Reactivity data in Table 13 confirm the qualitative observations that PAH generally display similar reactivities toward ozone and that alkylation enhances this reactivity considerably. The data suggest that in urban water supplies treated with ozone, lifetimes of PAH would be quite short only if the ozone were not consumed more rapidly by other organics and organisms or if the ozone did not evaporate.

A very recent report (Hoigne and Bader, 1975) suggests that the active species in reactions of ozone with organic compounds in water is HO• radical. Although extremely reactive this radical is produced from ozone slowly at rates which correspond closely to those estimated from Il'nitskii's data.

Degradation of PAH in Air

Each thousand cubic meters of urban air contains several micrograms of PAH adsorbed on particulate (Table 8) and as shown by Pupp et al. (1974) for some PAH, equal or greater amounts also may be found in the vapor phase (Table 6). No data are reported for chemical reaction of PAH in the vapor phase, but a number of observations concerning the decomposition of PAH, particularly benzopyrene adsorbed on particulate

Table 13

RATE CONSTANTS FOR REACTION OF PAH WITH OZONE IN WATER AT 25°C [a]

PAH	k_2, l mol^{-1} sec^{-1}	$t_{1/2}$, min [b]
Pyrene	170	41
Benz(a)pyrene	110	63
Benz(a)anthracene	260	27
Dimethylbenzanthracene	>680	<10
Dibenzanthracene	280	25

[a] Il'nitskii 1968

[b] Calculated from $t_{1/2} = 0.69/k_2 [O_3]$; $[O_3] = 10^{-4}$ M.

in air, summarized in the NAS report (1972), point to rapid decomposition with half-lives of hours owing to reactions with singlet oxygen, ozone, or other constituents of urban smog. Particular emphasis is given in the NAS report to the enhanced reactivity of PAH adsorbed on some surfaces such as silica or alumina where radical cations form readily and react to give oxygenated products, even in the absence of light. No data exist from which to calculate reliably lifetimes of PAH on suspended particulate, but some data exist from which lifetimes in the vapor state may be estimated. It is likely that rates of equivalent reactions on particulates will be slower.

The two principal oxidizing species in urban air are HO• radical, formed through a cycle involving photolysis of NO_2, water, CO, and simple organic compounds (Hecht 1974), and ozone formed from the O atom and oxygen. Recent measurements of HO• radical concentrations in urban air give values of 10^{-14} M (Niki 1975); ozone concentrations vary in clean air and are about $2 \cdot 10^{-9}$ M, but may be ten times larger in polluted air (Levy 1971).

Rate constants for reaction of PAH with HO• radical are not known; if we assume that the reactivity of PAH is similar to that of ethylene which is nearly diffusion controlled (Wilson 1971), $k_2 = 10^{9.3}$ l mol^{-1} sec^{-1} and for a pseudo-first order reaction

$$t_{1/2} = 0.69/k_2[\cdot OH] = 0.69/10^{9.3} \cdot 10^{-14}$$

$$t_{1/2} = 9.6 \text{ hr.}$$

This average half-life value for PAH is based on the largest reasonable rate constant for reaction with HO• radical; therefore, it is probable that true half-lives are longer.

Reactions of PAH with ozone in the vapor phase have half-lives shown in Table 14. These data are based on rates in water calculated from the

Table 14

HALF-LIVES FOR REACTIONS OF PAH WITH OZONE IN THE GAS PHASE

PAH	k_2 [a] $l\ mol^{-1}\ sec^{-1}$	$t_{1/2}$ hrs [b]
Pyrene	170	560
Benzo(a)pyrene	110	870
Benz(a)anthracene	260	368
Dibenzanthracene	280	342
Dimethylbenzanthracene	>680	>141

[a] See Table 13

[b] $t_{1/2} = 0.69/k_2 2 \cdot 10^{-9}$

data of Il'nitskii et al. (1968) given in Table 13, and a steady state concentration of ozone in the vapor phase of $2 \cdot 10^{-9}$ M (Levy 1971).

These values for $t_{1/2}$ are so large that reaction with ozone hardly seems likely to be an important process for conversion of PAH. The same conclusion applies to PAH adsorbed on particulate unless the PAH are greatly activated by adsorption.

Reactions involving singlet oxygen with simple organic compounds in the vapor phase are thought not to be important in smog chemistry owing to its very low concentration and the variety of processes that rapidly quench it (Dermerjian 1974).

However, the self-sensitized photooxygenation of some PAH involves intimate contact between PAH and singlet oxygen, which greatly increases the probability of further reaction before singlet oxygen is quenched (Stevens 1968, Algar 1970). Thus, the data of McGinnes and Snoeyink (1974), summarized in Table 11, are likely to be relevant to gas phase processes as well, with half-lives of 2-14 hours under similar conditions of light flux.

The oxygen atom, produced in the air by photolysis of NO_2 and ozone, is thought to play a minor role in oxidation of organic compounds; its ambient concentration is extremely low (Jaeger 1973, 1974), and it seems safe to assume that it cannot have an important role to play in removing PAH either from the vapor phase or from particulate.

Other reactants in urban atmospheres and in combustion plumes include nitrogen oxides and sulfur dioxide. Although the NAS report (1972) implies that NO_2 might react with PAH via a facile electrophilic substitution reaction, it is doubtful that such a reaction is fast enough to be of any importance under atmospheric conditions where NO_x concentrations are near 10^{-7} M. At higher temperatures and with NO_x concentrations closer to 10^{-5} M, as found in combustion plumes, such reactions become more probable. Sulfur dioxide has recently been implicated in reactions with PAH on particulate from chimneys (Jaeger 1973, 1974). In the

laboratory, benzopyrene and pyrene, adsorbed on alumina, react with SO_2 fairly rapidly under some conditions, possibly through a radical cation intermediate, to produce sulfonic acids (Jaeger 1973).

Toxicity, Bioaccumulation, and Biodegradation

Much of the information on the toxicity, accumulation, and degradation of the polycyclic aromatic hydrocarbons (PAH) in biological systems has been discussed and summarized in a comprehensive treatise published by the National Academy of Sciences (NAS, 1972). This work reviews pertinent reports published up to 1971 and should be consulted for information regarding toxicity in terms of carcinogenicity, teratogenicity, and mutagenicity, accumulation in plants and mammals, and degradation (metabolism), particularly by mammalian systems, of PAH compounds.

The NAS report does not include information on the non-oncogenic, -mutagenic or -teratogenic effects of PAH compounds in mammalian, non-mammalian, or plant life, nor does it address such topics as the accumulation of these compounds in wild life (animals, plants, and protists) or the degradation of the compounds by organisms other than laboratory mammals and isolated enzyme systems.

Toxicity

Little is known about the toxicity of PAH compounds aside from information that many are carcinogenic in laboratory mammals and in some cases, humans, and that some have been shown to be mutagens or teratogens.

Algae

Table 15 summarizes acute toxicity data obtained by Fitzgerald and coworkers (1952) for various species of algae exposed for 24 hours to phenanthroquinone, which is a degradation product of phenanthrene.

Table 15

ACUTE TOXICITY OF PHENANTHRAQUINONE TO BLUEGREEN ALGAE

Algae Species	Percent Survival				
	Phenanthraquinone Concentration (μg/l)				
	0	40	80	120	800
Microceptis aeroginosa	100	75	5	0	0
Microceptis incerta	100	0	0	0	0
Anabaena circinalis	100	0	0	0	0
Glocotrichia echinulata	100	25	0	0	0
Aphanizomenon flos aquae	100	0	0	0	0
Anaceptis marina	100	0	0	0	0
Coccochloris peniocystis	100	-	5	0	-
Gloecapsa membranina	100	100	100	50	10
Gloecapsa dimidiata	100	100	100	100	10
Gloecapsa alpicola	100	100	50	50	0
Plectonenia nostocorum	100	50	50	25	10
Nostoc muscorum	100	100	100	100	0
Nostoc commune	100	100	100	100	0
Calothrix parietina	100	10	10	10	0
Chlorella pyrenoidosa*	100	100	10	0	0

*Green alga

Source: Fitzgerald, et al., 1952.

The data indicate that with the exception of Anaceptis marina and Coccochloris peniocystis the algae that tend to produce noxious blooms (the first six) are more sensitive to phenanthraquinone than algae that do not produce blooms (last nine).

At low concentrations (10-20 μg/1), fluoranthrene, 1,12-benzoperylene, 3,4-benzofluoranthene, indeno(1,2,3,cd)pyrene, 1,2-benzanthracene, 3,4-benzopyrene, and 1,2,5,6-dibenzanthracene are reported to promote the growth of Chlorella vulgaris, Scenedesmus obliquus, and Ankistiodesmus oranunii (Graef and Nowak, 1966). The growth promoting potency appeared to correspond to carcinogenic potency. Benzo(a)pyrene was most effective; fluoranthene and 1,12-benzoperylene were least effective.

Higher Plants

It appears that photooxidation products of some PAH compounds are more toxic to leafy plants than the parent compound. Halbwachs and Hlabwatsch (1968) found that acridine, anthracene, fluoranthene, 9-methylanthracene, and 9,10-dihydroanthracene were toxic to higher plants only when the fumigated plants were exposed to direct sunlight. Chrysene, fluorene, phenanthrene, and carbozole were without this effect. 2-Methylanthracene was phytotoxic with or without direct sunlight. Plants sprayed with antioxidants were protected against the phytotoxic effects of those PAH compounds that were toxic only in direct sunlight. This effect could result from either preferential light absorption or from chemical interference with oxidation.

Although some PAH compounds have been shown to be toxic to plants, Graef and Nowak (1966) found that application of 10-20 μg/1 fluoranthene, 1,12-benzoperylene, 3,4-benzofluoranthene, indeno(1,2,3, cd)pyrene, 1,2-benzanthracene, 3,4-benzpyrene, or 1,2,5,6-dibenzanthracene to cultures of tobacco, rye, or radish promoted their growth, and as they observed with algae (see above). The degree of effectiveness in promoting plant growth appeared to correspond with increased carcinogenic potency.

Bacteria

Although a variety of microorganisms can metabolize and thus degrade various PAH compounds, PAH compounds may be toxic at high concentrations. Hass and Applegate (1975) reported that at concentrations of 10^{-7} to 10^{-5} M, anthracene, phenanthrene, chrysene, 1,2,3,4-dibenzanthracene, and pentacene inhibited the growth of cultures of Escherichia coli. On the other hand, 1,2-benzanthracene, 1,2,5,6-dibenzanthracene, and 3,4-benzpyrene, stimulated bacterial growth at these concentrations; and tetracene and pyrene had little or no effect. On the basis of this information the authors concluded that utilization of PAH compounds for growth by E. coli requires an angular acene molecule. PAH compounds with linear acene structures tend to inhibit growth. Inspection of the structural formulas of these compounds will show that this conclusion is erroneous.

In a series of studies on the oxidation of organic chemical carcinogens by activated sludge, Malaney and coworkers (1965, 1966, 1967) found that many PAH compounds were toxic to sludge microorganisms (as measured by O_2 uptake), and that sludge microorganisms from different sewage treatment plants reacted differently to the compounds.

Although 500 mg/l was the only concentration used, the tests were conducted with 2500 and 5000 mg/l suspended solids to determine the effect of doubling the bacterial population on oxidation. Doubling the bacterial population appeared to decrease the toxicity and to a certain extent, increase oxidation.

Listed below are the compounds that were toxic when the suspended solid concentration was 2500 mg/l.

1,2,5,6-dibenzanthracene

7-methyl-1,2-benzanthracene

1,2,4,5-dibenzpyrene

3-methylcholanthrene

2-nitrofluorene

2-fluoreneamine

N-2-fluorenylacetamide

7,9-dimethylbenz(c)acridine

7,10-dimethylbenz(c)acridine

Dibenz(a,h)acridine

Dibenz(a,j)acridine

Studies by Joyce and White (1971) showed that Staphylococcus aureus developed an electron transport system whenever the growing cells were aerated. This occurred simultaneously with increases in phospholipids and carotenoids. Addition of 3,4-benzopyrene to the culture system slowed the formation of the electron transport system, inhibited cytochrome oxidase synthesis, and depressed the synthesis of phospholipids and carotenoids. In earlier work, White (1970) found that the growth of S. aureus was inhibited by 10^{-5} M benzo(a)pyrene, benzo(e)pyrene, and dibenz(a,j)acridine as well as by the 2-ringed aromatic hydrocarbons such as α-naphthylamine and β-naphthylamine.

Invertebrates

Exposure to 0.5% solutions of 3,4-benzopyrene, 3-methylcholanthrene, or dimethylbenzanthracene for several weeks resulted in the formation of hyperplasia and incipient tumors in the earthworm (Lumbriculus terrestris) (Gersch 1954). Planaria treated with 3,4-benzopyrene or 3-methylcholanthrene developed lethal growths upon forced regeneration, and offspring from the treated animals developed lethal papilliform tumors (Foster 1969). Tumors have also been reported in snails treated with 3,4-benzopyrene (Krieg 1970).

According to Epstein and coworkers (1963), 3,4-benzopyrene is lethal to the unicellular invertebrate Paramecium caudatum when its administration is followed by exposure of the animal to sublethal levels of ultraviolet light. This response is inhibited by the presence of anti-oxidants. This suggests that some photooxidation product of benzopyrene is the toxic factor and supports the previously described results on the effects of photoirradiation on the phytotoxicity of certain PAH compounds.

In sponges, 500 mg of 3,4-benzopyrene per 100 ml of seawater caused choanocyte damage and abnormal growth of the oscular tube (Korotkova and Tokin, 1968). This effect was observed only in the highly colonial sponges such as Leucosolenia complicata and L. variabilis, and not in the simple spongelike Sycon raphanus. Injections of 3,4-benzopyrene are reported to increase ciliary activity and metabolism in fresh water mussels (Haranghy 1956).

In the housefly (Musca domestica) Cantwell and coworkers (1966) reported that exposure to 2,7-diaminofluorene, N-fluoren-2-yl-acetamide, 7-fluorofluorene-2-acetamide, and N-fluoren-2-yl-N-hydroxyacetamide inhibited growth, pupation, and adult emergence.

Fish and Amphibians

Little is known about the toxicity of PAH compounds in fish and amphibians. Compounds for which there is some toxicological information are anthracene, sodium anthraquinone-α-sulfonate, chrysene, phenanthrene, and phenanthraquinone.

Screening studies were performed by Applegate, et al., (1957) on a large number of different chemicals as part of a program to identify a toxicant specific for lamprey. The highest concentration employed was 5 mg/l, and the maximum exposure time was 24 hours. They reported that anthracene and chrysene had no effect on the rainbow trout, bluegill sunfish, or larval lampreys. Phenanthrene was lethal to rainbow trout

and bluegill sunfish at 5 mg/l in 12 hours, but had no effect on lamprey larvae. Other investigators (McKee and Wolfe 1963) report lethal concentrations of 1 to 5 mg/l for phenanthrene. In the mosquito fish, the 96-hour TL50 for phenanthrene is reported as 150 mg/l (EPA 1970).

Phenanthraquinone was nontoxic to the black crappie (Pomoxis migromaculatus), emerald shiner (Notropis atherenoides), blunt nose minnow (Hyborhynchus notatus), rock bass (Ambloplites rupestris) or large mouth bass (Micropterus salmoides) when they were exposed for 48 hours to solutions containing excess compound (Fitzgerald, et al., 1952).

Manfred (1970) reported that painting of 20-30 mg of benzopyrene and 20 mg of methylcholanthrene on the skin of short-lived fish (Rhodeus amarus and Gasterosteus aculeatus) for 2 to 7 months produced epitheliosis. Injection of benzopyrene produced injection site necrosis, but no neoplasms. No effect was observed in a similarly treated long-lived fish (Cyprinus carpio). When fed at a rate of 0.3 μg/mg body weight for 110 days, 3,4-benzopyrene and 3-methylcholanthrene increased the rate of respiration in the fish Platypoecilus maculatus and Xiphophorus helleri by 15 to 30% over controls. This treatment caused Xiphophorus helleri to grow about 25% slower than controls; however, in Platypoecilus maculatus, both compounds enhanced the rate of growth 2.0 and 2.5-fold, respectively.

The low lethal dose for 3,4-benzopyrene is reported to be 11 mg/kg in the frog (NIOSH 1974). This compound is also reported to induce tumors in amphibians (Leone 1953; Seilern-Aspang, 1962, 1963; Balls 1964) and to cause abnormal morphological development in tadpoles (Dontenwill 1953, DeLustig 1971, Matos 1973).

On the other hand, Breedis (1950) did not observe tumor formation in salamanders injected subcutaneously with 3,4-benzopyrene; and Arffman and Christensen (1961) reported that administration of this

compound as well as dibenz(a,h)anthracene and 3-methylcholanthrene to a species of newt produced epithelial proliferation but no tumors. Of the three, dibenzanthracene was the most potent.

Application of 1,2,5,6-dibenzanthracene or 3,4-benzopyrene to the amputated tail of the newt, Triturus viridescens, failed to produce tumors; however, the rate of tail regeneration was markedly reduced by these compounds (Pizzarello 1966). Lecamp and Delsol (1947) reported also that 3,4-benzopyrene did not induce tumors in regenerating limbs of the accoucheur toad but inhibited the rate of regeneration and appearance of the formed limb. Similar results were observed by Prada (1946) in Triton vulgaris.

Matoltsky (1947) reported no tumor formation in the amphibians, Rana esculénta and Triton cristatus injected with an 0.3% solution of 3,4-benzopyrene. However, he observed hemorrhaging in the kidney and liver, parenchymal degeneration, fatty degeneration, and necrosis. In the frog (R. esculenta) he observed pulmonary edema, cellular infiltration, and edematous swelling of the alveolar walls as well as edema of the skin and abdomen. Bonte (1950) also failed to induce tumors in frogs with 3,4-benzopyrene, implanted or painted on the skin; however, the compound produced atrophy and regression of the mucous glands of the skin and increased the permeability of the skin to water.

Dontenwill (1953) observed inhibition of cleavage and disturbances in the formation of blastomeres and neurolation in Triton and Axototl eggs exposed to unspecified concentrations of 3,4-benzopyrene. Colombo (1948), however, found that 3,4-benzopyrene concentrations of 1:2000 to 1:20,000 had no effect on the development of the ova, morula, or gastrula of the frog, Rana esculenta. Ruhland (1954) observed that 1:1000 to 1:10,000 solutions of 3,4-benzopyrene reduced the motility of the sperm of Rana fusca; however, eggs fertilized by these sperm developed normally.

Birds

Intratracheal administration of 3-methylcholanthrene (dose not specified) to ducks produces acute and chronic inflammation, and prolonged administration of the compound produced a variety of pulmonary tumors (Rigdon 1961). Administration of 3,4-benzopyrene also produces chronic pulmonary inflammation, but no tumors (Rigdon 1965b). Benzopyrene does not appear to be acutely toxic to ducks or chickens given a single oral dose of 250 mg (Rigdon 1963a).

Administration of up to 2.5 mg benzopyrene/g of food for 24 days does not affect the growth or survival of chicks, nor does a diet of 0.1 mg/g of food have any effect on the sperm, ova, egg fertility, or chicks from eggs obtained from treated hens (Rigdon 1963b).

Mammals

Although many PAH compounds have been tested for carcinogenicity, there appears to be little information on the acute and subacute toxicity of these compounds. Studies concerned with effects of PAH compounds on enzyme systems and other biochemical factors have usually been aimed at elucidating the mechanism of carcinogenic action. Some of these studies were reviewed because the information relates to the effects of PAH compounds on metabolic processes.

In an *in vitro* study on the effect of 5 PAH compounds on the activity of selected enzymes, Gemant (1967) observed that the activity of catalase, an enzyme that acts on hydrogen peroxide and thus regulates the amount of this compound in tissues, was reduced by up to 50% by the PAH compounds. In order of decreasing inhibition potency, the compounds were 3-methylcholanthrene, 3,4-benzopyrene, 1,2-benzanthracene, 9,10-dimethyl-1,2-benzanthracene, and anthracene, which suggests that the

catalase-inhibiting potency of the PAH compounds is related to their carcinogenic potency. None of these compounds had any effect on the activity of peroxidase.

The activity of lipoxygenase, another oxidoreductase, was only 44% of the control level in the presence of 3,4-benzopyrene, the most potent inhibitor, and only 46 and 51% of the control value in the presence of methylcholanthrene and dimethylbenzanthracene. Of the three carcinogens, dimethylbenzanthracene inhibited that activity of ribonuclease most and benzopyrene was least effective. None of the carcinogens had any effect on the activity of trypsin.

In mice injected with 1.25 mg of 3,4-benzopyrene or 2.5 mg of 3-methylcholanthrene (Draganov 1966) a significant increase occurred in succinic dehydrogenase activity in lung tissue 60 days after injection but not at 30 days after the injections. He reported that enhanced succinic dehydrogenase activity occurred at the same time that pathological changes in the lung tissue were observed. Zinnari (1964) observed that same effect in liver tissue, but enhanced succinic dehydrogenase (SH) activity occurred much more rapidly. In mice injected with a 2% solution of 3,4-benzopyrene, the activity of succinic dehydrogenase increased about 3-fold above the control level on the second day and gradually decreased to near control levels by the 30th day. However, the degree of morphological changes in the liver mitrochondria increased with time, rather than with enhancement of succinic dehydrogenase activity.

The effect of PAH compounds on the activity of cathepsin from subcellular fractions of rat liver homogenates was investigated by Lomsadze and coworkers (1969). They found that dimethylbenzanthracene, 3-methylcholanthrene, 1,2-benzopyrene, and anthracene lowered the activity of the

enzyme at a concentration of 0.005M. Anthracene was least effective. These investigators also found a reduction in cathepsin activity in the rat liver 2 to 5 months after a single injection of 5 mg of dimethylbenzanthracene to rats.

DeLuca (1969) reported no changes in liver glutamic-oxalacetic transaminase (GOT) or glucose-6-phosphate activity in rats injected with 0.1 mg 3,4-benzopyrene; however, at a dose of 0.5 mg, the activity of GOT was enhanced.

Intravenous injection of 0.5 mg of anthracene, pyrene, perylene, 3,4-benzopyrene, or 1,2,5,6-dibenzanthracene caused an increase in liver SH levels in mice within 15 minutes to 1.5 hours. Subsequently, the SH levels dropped to below normal.

Rigdon (1965a) observed a decrease in weight of mice fed up to 1.0 mg 3,4-benzopyrene/g of food; however, he found that the decrease was due to a decrease in food intake rather than to benzopyrene toxicity. Reduced intake was due to the ability of the animals to detect the presence of the compound in partial rejection of the treated food. Mice reared on treated food from the time of weaning readily ate the food and gained as much weight as the control animals. Rigdon (1966) observed that mice from mothers fed 3,4-benzopyrene during pregnancy and lactation did not grow as rapidly as the controls beginning 10-12 days of age. This effect was attributed to nutritional deficiencies rather than to a direct effect of benzopyrene.

Bioaccumulation and Biodegradation

A relatively large number of PAH compounds have been identified in living matter. Data from field and laboratory studies indicate that organisms throughout the phylogenetic scale can take up PAH compounds from the environment, including food, and also metabolize these compounds.

The metabolism of PAH compounds by mammals has been the object of much research. Ample evidence exists that these compounds are metabolized by mammals and some evidence that the products of metabolism may be the carcinogenic factor for some of the compounds. The metabolism of PAH compounds by mammals is well reviewed in the NAS document (1972) as is their accumulation by plants; hence, neither subject will be discussed to any great extent in this report.

Bacteria

Although measurable quantities of PAH compounds have been found in bacteria grown in PAH-contaminated media, and a number of studies have shown that bacteria metabolize certain PAH compounds, it is not clear whether the compounds studied are adsorbed or absorbed by the bacteria or whether metabolism occurs intra- or extra-cellularly.

When adapted to soil containing 3,4-benzopyrene, Pseudomonas aeruginosa and Escherischia coli took up about 90% of the compound from the medium, metabolized 10 to 26%, showed enhanced growth, and contained 3 to 15 times more protein than normal (Lorbacher, et al., 1971). Studies by Moore and Harrison (1965) showed that various enterobacteria such as Salmonella typhimurium, Aerobacter aerogenes, and Escherischia coli as well as various strains of Saccharomyces cerevisiae are capable of accumulating 3,4-benzopyrene; however, they metabolized little of the compound. Uptake in E. coli amounted to 10^{-10} to 2×10^{-10} µg benzopyrene per cell.

In a series of reports, Poglazova (1971) and Poglazova and coworkers (1966, 1967a,b, 1971), observed uptake of 3,4-benzopyrene by up to 20 strains of soil bacteria, including Mycobacterium flavum, M. rubrum, M. lacticolum, M. smegmatis, Bacillus megaterium mutilate, and Bacterium sphaericus. M. rubrum and M. flavum metabolized about 50% of the compound in 4 days. 3,4-Benzopyrene is also taken up and metabolized by Endomyces

magnusii and by Candida lepolytica (Petrikevich, et al., 1964), and is accumulated from forest soil compost by Clostridium putrifaciens (Mallet 1965).

Shabad (1968) and coworkers (1971a,b) studied the rate of 3,4-benzopyrene destruction by various strains of soil bacteria isolated from various areas and found that strains isolated from soil highly contaminated with benzopyrene were capable of metabolizing from 75 to 86% of the compound in 5 days, while those from low benzopyrene areas could metabolize only 48 to 59% in the same period of time.

Bacteria also metabolize anthracene and phenanthrene (Evans, et al., 1965), 9,10-dimethyl-1,2-benzanthracene, 1,2,5,6-dibenzanthracene, 1,2-benzanthracene, 1,2-benzopyrene, pyrene, 1,12-benzperylene, and perylene (Fedoseeva, et al., 1968). The last 7 compounds were metabolized by Bacillus megaterium at the same rate, regardless of concentration or solubility in the medium (Fedoseeva, et al., 1968). Soil microorganisms also appear capable of metabolizing 3-methylcholanthrene (Lijinsky 1956).

Aquatic bacteria appear to be less efficient in metabolizing PAH compounds than soil microorganisms. Bacteria in power plant and coke oven wastewater contaminated with 3,4-benzopyrene metabolized less than 15% of the compound (Poglazova, et al., 1972). Malaney (1966) reported that anthracene was only slightly oxidized by sewage sludge bacteria acclimated to benzene.

Malaney and coworkers (1967, See also Lutin 1965) studied the susceptibility of 17 PAH compounds to oxidation of activated sludge microorganisms and found that most were resistant to oxidation. Phenanthrene, a tricyclic compound, was most susceptible to oxidation. For this compound the oxygen uptake by the sludge amounted to 22 to 46% of

the theoretical oxygen demand (TOD) calculated on the basis of complete oxidation of the compound. In contrast, oxygen uptake for the two-ringed compound, naphthalene, amounted to about 33 to 64% of TOD. Another tricyclic compound, anthracene, was very resistant to oxidation (2 to 13% of TOD). Of the remaining compounds, the quadracyclic compound, 9,10-dimethylbenzanthracene was most susceptible to oxidation (19.6% of TOD); however, the other benzanthracenes were resistant. The penta- and hexacyclic compounds were essentially inert. The acridines were also inert, and some were toxic. Some of the fluorenes were susceptible. The compounds that were tested are listed below:

(1) 9,10-Dimethylanthracene
(2) 7-Methyl-1,2-benzanthracene
(3) 9,10-Dimethyl-1,2-benzanthracene
(4) 1,2-Benzanthracene
(5) 1,2,5,6-Dibenzanthracene
(6) 3,4-Benzopyrene
(7) 1,2,4,5-Dibenzopyrene
(8) 20-Methylcholanthrene
(9) 2-Nitrofluorene
(10) 2-Fluoreneamine
(11) N-2-fluorenylacetamide
(12) 7,9-Dimethylbenz(c)acridine
(13) 7,10-Dimethylbenz(c)acridine
(14) Dibenz(a,h)acridine
(15) Dibenz(a,j)acridine
(16) Anthracene
(17) Phenanthrene

It thus appears that susceptibility to microbial degradation is not necessarily a function of the number of benzene rings in the structure of the compound.

Marine bacteria are capable of metabolizing phenanthrene, anthracene, and fluorene (Dean-Raymond 1975).

Higher Plants

A variety of PAH compounds have been found in plants. These include:

Anthracene	3,4-Benzopyrene
1,2-Benzanthracene	Chrysene
1,2,5,6-Dibenzanthracene	Coronene
10,11-Benzfluoranthene	Fluoranthene
11,12-Benzfluoranthene	Indeno(1,2,3,c,d)pyrene
1,2-Benzoperylene	Perylene
1,12-Benzoperylene	Phenanthrene
1,2-Benzopyrene	Pyrene

Reports that we have reviewed indicate that PAH compounds may enter plants through the leaves and roots; however, whether the roots or the leaves constitute the major route of entry is not clear.

Shabad (1968) analyzed the leaves of a variety of plants surrounding an oil refinery and discovered that the 3,4-benzopyrene content of the leaves diminished with the distance away from the refinery. Broad, prostrate leaves tended to contain larger amounts of the compound than slender, upright leaves, and washing removed a significant amount of the compound. He concluded that the source of 3,4-benzopyrene in the leaves was atmospheric fallout.

Shabad and coworkers (1971a) also grew plants (nasturtiums and asters) in a 3,4-benzopyrene-treated nutrient medium and found the compound distributed throughout the plants, indicating root absorption. Similar results were obtained by Doerr (1965) with peas, wheat, and barley grown in 3,4-benzopyrene-treated soil and nutrient media.

Little is known about the metabolism of PAH compounds by plants. Shabad and Cohen (1972) stated that Durmishidze (1968) demonstrated that vascular plants are capable of metabolizing hydrocarbons, including those of cyclical structure.

Aquatic Organisms

Little information is available on the uptake or metabolism of PAH compounds by aquatic organisms. 3,4-benzopyrene has been found in marine algae, plankton, molluscs, and worms (Mallet 1967). It and perylene have been measured in phyto- and zooplankton, higher algae forms, and crustacea (Niaussat and Auger, 1970a,b). Niaussat and Auger (1970a,b) also reported that the biota in a lagoon contaminated with 3,4-benzopyrene and perylene contained both compounds. The water contained 1.6 and 3.05 μg/l of benzopyrene and perylene, respectively. Plankton contained 0.73 and 0.27 μg/100 g (7.3 and 2.7 μg/kg), the higher algae contained ~~576~~ 376 and ~~865~~ 716 μg/kg, and isopod crustacea contained 536 and 865 μg/kg, indicating that accumulation and perhaps biomagnification occurs. Crustaceans collected from the ocean outside the lagoon contained little or no benzopyrene or perylene.

Plankton collected in the North Atlantic were analyzed for fatty acid content and 3,4-benzopyrene by deLima-Zanghi (1968). Plankton collected from coastal areas contained significant amounts of benzopyrene, whereas those collected from the high seas were uncontaminated. No correlation was found between lipid content or type (saturated or unsaturated) and benzopyrene content.

Freshwater worms of the genus Tubifex exposed to 0.01, 0.1, and 100 μg 3,4-benzopyrene /liter for 6 to 11 days, accumulated up to 88.2 mg/kg of the compound. The amount accumulated increased with increasing exposure concentrations. When placed in uncontaminated flowing water for 40 days, the worms lost about 75% of the compound (Scaccini-Cicatelli 1966).

That mammals can metabolize at least some PAH compounds is well-known. There is evidence that chickens and ducks also can metabolize 3,4-benzopyrene (Rigdon 1963a).

Biodegradation Products

As mentioned previously, the metabolism of PAH compounds and the identification of their metabolites is known primarily from bacterial and mammalian systems. The metabolism of PAH compounds by plants, invertebrates other than bacteria, and by the lower vertebrates is unknown.

In mammals the major metabolites of PAH compounds are hydroxylated derivatives, and carboxylic acid derivatives are also formed (Sims 1970). A review of current knowledge on the metabolic products of PAH compounds in mammals is given in the NAS document (NAS 1972). In general, mammals and perhaps birds do not have the ability to degrade PAH compounds completely. Some of the metabolites have been found to be active carcinogens but less so than the parent compound (Boyland and Sims 1967) or mutagens (Cookson, et al., 1971; Ames, et al., 1972). These metabolites include the hydroxylated derivatives as well as the epoxides. As a rule the parent compound as well as the metabolites is excreted via the urine and to a certain extent, the feces (Evans, et al., 1965).

Bacteria have been shown to utilize PAH compounds as a carbon source for growth, and evidence exists that they can metabolize PAH compounds much more completely than do mammals. This evidence comes from studies on only a few PAH compounds, particularly anthracene and phenanthrene.

According to Evans and coworkers (1965), phenanthrene is metabolized by soil pseudomonads to 1,2-dihydroxynaphthalene via several steps involving intermediates such as trans-3-4-dihydro-3,4-dihydroxyphenanthrene (Colla, et al., 1959), 3,4-dihydroxyphenanthrene, and

Marine fish absorb 3,4-benzopyrene through the gills, metabolize the compound in the liver, store it and its metabolites in the gall-bladder, and finally excrete both in the urine (Lee, et al., 1972).

It thus appears that at least some PAH compounds are accumulated by a variety of aquatic organisms; however, so far the metabolism of these compounds has been demonstrated only in bacteria and fish.

Birds and Mammals

We did not find any report on concentrations of PAH compounds in other than experimental animals.

Gorelova and associates (1971) reported only a trace and, in many cases, no detectable levels of 3,4-benzopyrene in the muscle, fat, liver, or blood of rabbits, pigs, cows, chickens, or ducks, or in the milk of the mammals or eggs of the birds that were given an unspecified amount of the compound in the diet for up to one year. Cherepanova (1971) fed the same kind of animals up to 10,000 μg of 3,4-benzopyrene per day for an unspecified time and found benzopyrene levels of no more than 0.26 μg/kg in the muscle, fat, and liver. Eggs contained no more than 0.007 μg/egg, and milk contained no more than 0.01 μg/liter.

When applied to the skin of mice or rats, known carcinogenic PAH compounds penetrate the skin more readily than non-carcinogenic ones, and are eliminated more slowly from the body. Grimm and Oehlert (1966) came to these conclusions in a study using radiolabeled 1,2,3-dibenz-anthracene and 1,2-benzopyrene, both of which are non-carcinogens, and 3-methylcholanthrene and 3,4-benzopyrene, both of which are known carcin-ogens. They found no difference in the distribution of either type of compound in the animals; but observed that radioactivity in rat skin declined at a faster rate than in mouse skin. Accumulation of other PAH compounds does not appear to have been studied.

1-hydroxy-2-naphthoic acid. 1,2-Dihydroxynaphthalene is then metabolized to cis-o-hydroxybenzalpyruvate (Davies and Evans 1964).

Evans and coworkers (1965) also proposed that anthracene is metabolized by soil pseudomonads to 2,3-dihydroxynaphthalene via several intermediates, including trans-1,2-dihydro-1,2-dihydroxyanthracene (Colla, et al., 1959), 1,2-dihydroxyanthracene, and 2-hydroxy-3-naphthoic acid, which is eventually metabolized to salicylate (Colla, et al., 1959; Rogoff and Wender 1957). The oxygenase responsible for the cleavage of all o-dihydroxyphenol derivatives appears to be catechol-2,3-oxygenase, a constitutive enzyme of Pseudomonas sp. (Evans, et al., 1965).

1-Hydroxy-2-naphthoic acid was also identified as a microbial metabolite of phenanthrene by Kaneko and coworkers (1968, 1969). They also reported (1969) that Pseudomonas is capable of metabolizing salicylic acid and catechol, which are considered products of phenanthrene and anthracene metabolism.

Biosynthesis

Graef (1966) reported 3 to 5 times more fluoranthene, 3,4-benzfluoranthene, indeno(1,2,3,cd)pyrene, 1,2-benzperylene, and 3,4-benzopyrene in beech, oak, and tobacco leaves that were turning yellow than in green leaves collected at the same time, and hypothesized that these polycyclic aromatic hydrocarbons are synthesized by the plants. To test this hypothesis, he grew rye, wheat, and lentils from seeds in a system free of 3,4-benzopyrene and found as much as 3.8 μg of 3,4-benzopyrene per 100 grams of plant sample after the seeds had sprouted. From this study he concluded that plants do indeed synthesize benzopyrene.

In a study by Hancock and coworkers (1970), plants along a railroad and from a control area were analyzed for anthracene, fluoranthene, pyrene, benz(a)anthracene, and benzo(a)pyrene. They found higher levels of these compounds in plants from the control area and, like Graef and Diehl (1966)

observed no seasonal change in benzopyrene-pyrene ratios in the leaves although they expected that if pyrene were from an exogenous source, it would undergo rapid photodegradation particularly during the summer, and thus cause a change in ratio. From these observations, they concluded that the PAH compounds found in the plants were synthesized.

Experiments by Borneff and associates (1968a,b) demonstrated that the alga, Chlorella vulgaris was capable of synthesizing fluoranthene, benz(a)anthracene, benz(b)fluoranthene, benzo(a)pyrene, benz(ghi)perylene, benz(k)fluoranthene, and indeno(1,2,3,cd)pyrene.

Although these studies strongly indicate that PAH compounds found in plants can be of endogenous origin, Grimmer and Duevel (1970) did not find any benzo(e)pyrene, benzo(a)pyrene, perylene, anthracene, benz(ghi)-perylene, dibenz(a,h)anthracene, or coronene in plants grown in greenhouses in which the air was supplied through special filters.

Biosynthesis of PAH compounds has been demonstrated in the bacteria Clastridium cultured in the presence of lipid extracts from marine plankton (Mallet, et al., 1967), in Bacillus badius cultured in the presence of lycopene, naphthelenic acid, and vitamin K (Niaussat, et al., 1970a), and in Welchia sp. (Brisou 1969).

Synthesis of PAH compounds by multicellular animals has not been demonstrated.

Biological Activity

The recent literature on the biological activity of PAH is rather heavily weighted toward carcinogenic and cocarcinogenic aspects. In accord with the limited objectives of this literature study, attention was not directed to such references unless they were specifically oriented toward structure-activity relationships or were obviously

pertinent to that subject. In contrast, the recent literature on PAH metabolism received much greater attention because of the current evidence that metabolic transformation is intimately involved with mechanisms of carcinogenicity and, therefore, with structure-activity considerations and other aspects of toxicity. For similar reasons, biochemical and immunological effects of PAH received special attention as did studies of absorption, distribution, and binding and reports of the biological effects of various transformation products of PAH.

Biological Effects

Many of the recent studies of the effects of PAH on sensitive receptors other than man have been concerned with differentiating between carcinogens and non-carcinogens. Examples include inhibition of RNA virus replication (deMaeyer 1964, Hsu 1966), stimulation of rat liver ribosomal protein and RNA synthesis (Hradec 1967), immunosuppression in the mouse (Stjernsward 1965, 1966), induction of chromosomal breaks (Rees 1970), enhancement of E. coli cell sensitivity to ultraviolet irradiation damage (Mirsov 1973), and growth promoting effects in plants (Graef and Nowak, 1966).

Biochemical studies of carcinogenic PAH indicate that these can inhibit a variety of plant, bacterial, and mammalian enzyme systems (Gemant 1967, Konstantinova 1973, Vysochina 1974, Lillich 1972) and repress tail regeneration in the newt (Pizzarello 1966, also p. 49 this report). Plant damage following application to leaves, however, appears to be due to products formed by photooxidation (Halbwachs 1968a,b). Interestingly, non-carcinogenic PAH may inhibit some effects of carcinogenic PAH (Hsu 1966).

Absorption, Distribution, and Binding

Absorption studies, involving primarily benzo(a)pyrene, have demonstrated uptake by microorganisms (Mallet 1965, Moore 1965), transplacental passage in mice (Shendrikova 1974) and inhibitory effects of soot and aerosol treatments on intratracheal absorption, removal, and elimination, with a resultant enhancement of carcinogenicity locally (Dontenwill 1968). Other absorption sites studied include mouse and rat skin (Grimm 1966, Sezaki 1963) and the rapid passage of PAH into lymph following intestinal absorption (Rees 1971, Mandelstam 1969). The absorption of various PAH is probably very similar although some differences between carcinogenic and non-carcinogenic compounds have been claimed (Grimm 1966). Furthermore, the uptake, distribution, metabolism, and binding to protein of one PAH such as benzo(a)pyrene can be altered by the presence of a second PAH such as phenanthrene or 3-methylcholanthrene (Flesher 1973, Anghileri 1967).

Binding of PAH to DNA appears to involve both intercalation (Kodama 1966, Craig 1970) and covalent bonding, which is dependent on metabolic activation (Kaufman 1973, Blackburn 1971); while binding of benzo(a)pyrene is enhanced by vitamin A deficiency. This can be counteracted by an inhibitor of the aryl hydrocarbon hydroxylase system, which reduces the ability of cells to metabolize PAH (Genta 1974). Alkyl substitution (7,12-dimethyl) appears to enhance DNA binding of benzo(a)anthracene (Yuspa 1970). While binding is also enhanced by ultraviolet irradiation of PAH, the significance of this method of activation to studies of relative carcinogenicity as a function of DNA binding is not clear (Blackburn 1971, Pascal 1971). Presumably, reactive intermediates of carcinogenic PAH are important in covalent bonding

since the final photooxidation products of benzo(a)pyrene seem not to be tumorigenic (Gubergrits 1974); photoirradiation studies demonstrate covalent bonding by 3,4-penzopyrene to DNA with no such bonding by non-carcinogenic 1,2-benzopyrene (Maevskii 1973).

Pertinent to carcinogenic activity and chronic toxicity is the fact that body fat absorbs large amounts of these hydrophobic compounds and they exhibit prolonged retention in fat, adrenals, and ovaries (Daniel 1967).

Physio-Chemical Correlates of Activity

Investigators have attempted to define physico-chemical (i.e., non-biological) phenomena which correlate theoretical chemical properties with carcinogenic or cytotoxic activities of PAH. These include positional reactivity toward ozonation (Moriconi 1968), free radical photogeneration or production in tissues (Okazaki 1971, Inomata 1972, Nagata 1966, Kotrikadze 1974, Rondia 1967), intermolecular electron transfer (Kavetskii 1966), one-electron oxidation to radical-cation intermediates and reaction of these with nucleotide bases (Wilk 1966, 1972), electrochemiluminescence (Kozlov 1967a, 1970; Mikhailovskii 1967), interactions with lipid monomolecular films (surface tension data) (Felmeister 1972), and semiconductor properties (Drost 1966).

Metabolism and Biological Mechanisms

A great deal of evidence exists linking biotransformation of PAH to their carcinogenic and cytotoxic properties. It is well known that the microsomal enzyme systems concerned can be induced or stimulated by a variety of drugs, insecticides, etc.; PAH are especially effective inducers, which substantially increase microsomal protein synthesis and alter rates of metabolism of endogenous substrates and PAH.

Considering the extremely low aqueous solubilities of PAH, biological transport probably depends on solubilization by albumin and other organic plasma and cellular constituents with a resulting facilitation of metabolism (Alvares 1970). Recent studies have demonstrated similar PAH metabolism by mouse fetal and placental tissues (Guibbert 1972), guinea pig alveolar and peritoneal macrophages (Tomingas 1971), Syrian golden hamster (Dontenwill 1968b), mouse embryonic fibroblasts (Belitskii 1970), and cultured human lymphocytes (Booth 1974) as well as by mice and rat-liver microsomes. In addition, there is ample evidence of metabolism by plants, microorganisms, and fish (pp. 53, 56, 57 this report).

PAH such as phenanthrene, benzo(a)pyrene, benzo(a)anthracene, and methylated analogs are metabolized by microsomal oxygenases to K-region epoxides followed by conversion by epoxide hydrase to dihydrodiols (Holder 1974, Sims 1971, Grover 1971a, Sims 1973a, Boyland 1965a), which are then conjugated with glutathione (Sims 1973a). Alkyl hydroxylation also occurs with methyl-PAH (Sims 1970, Gentil 1971).

Several recent studies indicate the importance of metabolic activation as a prerequisite for cytotoxicity, reaction with nucleophiles, macromolecular binding, and carcinogenicity (Gurtoo 1974, Diamond 1970, Huberman 1971, 1972, Borgen 1973, Sims 1973, Cavalieri 1974, Duncan 1970, Ahn 1974, Gelboin 1969, Aleksandrov 1974, Flesher 1970). In the case of methyl-PAH, increased carcinogenic activity may result from metabolic formation of hydroxymethyl derivatives (Boyland 1965b). Similar conclusions result from studies of microsomal enzyme pretreatment induction by benzo(a)anthracene and other PAH (Gelboin 1972). Several studies, however, also indicate that either pretreatment or cotreatment with various PAH can also decrease carcinogenic and cytotoxic effects of PAH by stimulation of metabolism (Conney 1966, Argus 1971, Levin 1967) or competitive inhibition of metabolism (Kunte 1969, Tomingas 1970a, 1970b); organophosphate insecticides also inhibit PAH metabolism (Weber 1974).

It appears that metabolic formation of epoxy and hydroxy intermediates is important in the cytotoxic and carcinogenic activities of these compounds, while rapid further degradation of these intermediates is biologically protective. The much greater covalent binding of 7,8-dihydroxy-7,8-dihydroxy-7,8-dihydrobenzo(a)pyrene (Borgen 1973) is indicative of this, as is the fact the benzc(a)anthracene-5,6-epoxide is much more active in malignant transformation of embryonic cells than is the parent hydrocarbon or its phenols or dihydrodiols (Huberman 1972).

Various aspects of PAH metabolism, including formation and metabolism of epoxide intermediates, are discussed in very recent reviews (Conney 1974, Wiebel 1974, Grover 1974).

Structure-Activity Relationships

During much of the past two decades the Pullman electronic theory of carcinogenic activity has dominated considerations of structure-activity relationships among PAH. This concept of reactive, electron-dense K-region double bonds and relatively inactive L-regions is still being considered in association with currently more popular ideas of metabolic activation as well as in its original terms. The theory remains useful in predicting carcinogenic activity in some series of unsubstituted PAH when competitive metabolism at the L-region is considered in conjunction with K-region reactivity (Scribner 1969). Since both carcinogenic and non-carcinogenic PAH can possess similar K-regions, it is evident that the simple idea that only the presence of this region is required for carcinogenicity is invalid (Cavalieri 1971). Theoretical calculations suggest that the fundamental theory retains some validity (Meyer 1969, Hoffmann, F. 1969), but fail to define any simple relationships between carcinogenic activity and K or L region reactivity indices (Sung 1972). Similarly, the relationships between carcinogenicity and

K and L region additivity with ozone are not clearly defined (Moriconi 1959, 1961, 1968). These difficulties are not surprising in terms of postulates that substitutions at different positions represent competing processes of activation for carcinogenesis and detoxification (Scribner 1973).

The potency of PAH as inducers of microsomal aryl hydrocarbon hydroxylase can be quantitatively described in terms of hydrophobic interactions, chemical reactivity, and the ability to participate in charge-transfer interactions; the critical step in both induction and carcinogenesis is considered to be the formation of a reactive K-region (Franke 1973). Additional considerations, however, are relative rates of metabolism and competing metabolism at K-regions and other loci (Sims 1970). K-region epoxides of PAH appear to be more active in cell transformation (Huberman 1972) and more reactive toward nucleic acid and protein fractions (Grover 1971b) than corresponding hydrocarbons, K-region dihydrodiols, and phenols. Appreciable differences exist in the rates at which these epoxides rearrange in neutral solution and are metabolized further (Swaisland 1973). Thus, in vivo indications of relatively low carcinogenic activity following administration of such epoxides (Boyland 1967) may not be indicative of the importance of these as active metabolites formed in vivo from the parent hydrocarbons. The importance of the epoxides is further emphasized by the fact that those derived from potent carcinogens are mutagenic in bacteria even though the parent hydrocarbons are not (Ames 1972, Cookson 1971).

Among PAH derived from petroleum cracking, carcinogenic potency is maximal in 4-5 ring compounds, largely benz(a)anthracene and its alkyl derivatives (Tye 1966a). Monomethyl derivatives of benz(a)anthracene are carcinogens of varying potency (Stevenson 1965, Roe 1972). These include the 6-, 7-, 8-, and 12-methyl derivatives, and various dimethyl

derivatives are also carcinogenic; positions 6, 7, 8 and 12 in benz(a)-anthracene are considered to form a "triangle of strong carcinogenicity" (Huggins 1967, Sugiyama 1973). The carcinogenicity of 4,5,10-trimethyl-benz(a)anthracene is especially high (Dunning 1968).

No simple correlation was found with regard to ethyl group position and carcinogenicity in substituted benz(a)anthracenes; 6,8- and 8,12-diethyl were potent carcinogens while 7,8- and 7,9-diethyl were inactive (Pataki 1972). Carcinogenic activity in benz(a)anthracenes appears to depend on at least one relatively flat surface and a geometric resemblance to nucleic acid base pairs (Pataki 1969).

Methyl substitution in appropriate positions appears to generally enhance PAH carcinogenicity. In chrysene the result is increased initiator potency in some cases, but 5-methylchrysene is a complete carcinogen (Hoffmann, D. 1974). In substituted benz(a)pyrenes mutagenicity decreases in the order 3,6-dimethyl > 3-methyl, 6-hydroxymethyl > benz(a)pyrene > 1,6-dimethyl (Fahmy 1973); carcinogenicity is in the order 6-methyl > benz(a)pyrene > 6-carboxaldehyde > 6-hydroxymethyl and appears to depend on either electron donor or acceptor properties (Dewhurst 1972).

Structure-activity correlations are claimed with respect to carcinogenicity and photodynamic action in Paramecium (Epstein 1964) and energy differences between lowest excited singlet and lowest triplet levels from spectroscopic data (Steele 1967).

Environmental Sources

Air

Polynuclear aromatic hydrocarbons are transported in the atmosphere adsorbed on particulates and bacteria. Their concentrations are roughly proportional to the amount of benzo(a)pyrene (BaP) and depend on the

temperature, the amount of sunlight, the traffic, and the geography of location. Urban concentrations tend to be around 10-100 times greater than nonurban. Olsen (1969) has found that the arithmetic mean for a nonurban location is 0.4 μg/1000 m^3 and for an urban location is 3 μg/1000 m^3. The figure of 0.4 μg/1000 m^3 can be assumed to be a low and safe level of benzo(a)pyrene in air. Winter concentrations tend to be greater than summer concentrations (IARC 1973). Table 16 lists the winter and summer concentrations for several cities in Europe and the United States. Location is specified when possible. The higher concentration in winter is probably due to lower rates of photooxidation in winter (Andelman 1970) and, especially, increased use of fossil fuel for winter heating.

PAH concentration is also influenced by the amount of automotive exhaust. The contribution due to traffic is not large, ranging from 5-42% (Sawicki 1967). Concentrations in Sydney, Cincinnati, and Detroit ranged as shown in Table 17 depending on the traffic. The BaP concentrations agree with another review's average BaP concentration of 6 μg/1000 m^3 for 100 U.S. cities (Sawicki 1967). Another source quoted the concentrations of the three compounds for six U.S. cities in terms of grams of organic atmospheric particulates (IARC 1973). Refer to Table 18 for these figures.

Water

Polynuclear aromatic hydrocarbons find their way to waterways adsorbed onto aerosols or bacteria (Andelman 1970). Although their solubility in pure water is essentially zero, they may exist in water in association with organic matter or colloids (micelles) as formed by synthetic detergents. An extensive review (Andelman 1970) on water listed the carcinogenic PAH concentrations in four types of water resources. These are listed in Table 19. Note that groundwater, in general, is least contaminated by PAH. This low level results from

Table 16

VARIATIONS IN PAH CONCENTRATIONS WITH SEASONS

	($\mu g/1000\ m^3$) Winter	($\mu g/1000\ m^3$) Summer	Ref.
Benzo(a)pyrene	0.6-104	0.03-4	(IARC 1973)
	26 (14 U.S. cities)	1.9 (14 U.S. cities)	(Olsen 1969)
Chrysene	20-361	2.5-3.6	(IARC 1973)
Benz(a)anthracene	94 (Siena)	1.6 (Siena)	(IARC 1973)
	361 (Bochun)	136 (Pittsburgh)	

Table 17

VARIATIONS OF PAH CONCENTRATION WITH TRAFFIC

	$\mu g/1000\ m^3$
Benzo(a)pyrene	2.5-6.5
Chrysene	1.8-13.3
Benz(a)anthracene	0.6-13.7

Source: IARC 1973

Table 18

PAH CONCENTRATIONS IN TERMS
OF TOTAL ORGANIC ATMOSPHERIC PARTICULATES

	Org. Atmos. (μg/gm) Particulates
Benzo(a)pyrene	110-670
Chrysene	150-490
Benz(a)anthracene	43-280

Ref: IARC 1973

Table 19

CARCINOGENIC PAH CONCENTRATIONS IN WATER SOURCES

Source	(μg/l)
Groundwater	0.001-0.1
Treated river and lake water	0.01-0.025
Surface water	0.025-0.100
Surface water, strongly contaminated	>0.100

Ref: Andelman 1970

filtration by soil profiles. As a point of reference, 0.017 μg/l of maximum permissible concentration of carcinogenic PAH has been suggested for human consumption (Andelman 1970). The World Health Organization has recommended a maximum of 0.2 μg/l PAH calculated as the sum of six easily identified compounds (Andelman 1970). The limit of 0.0075 μg/l for the BaP component of this total was recommended with total carcinogenic PAH limits of 0.03 μg/l.

Another review has broken down the concentration of three PAH in water (IARC 1973). These results are presented in Table 20. Note that drinking water concentrations, when added together, amount to much greater concentrations than those listed in Table 19. Considering that BaP is generally said to constitute between 1% and 20% of the total carcinogenic PAH, the figures in Table 20 are several magnitudes greater than the recommended allowable drinking water concentrations.

Contaminated waters can have seriously large PAH concentrations. Andelman (1970) has also quoted BaA concentrations of 0.025-10 μg/l and BaP concentrations of 0.001-1.84 μg/l in industrial and bitumen contaminated effluents. Water from households, trades, roads, and industrial sources had up to 31.4 μg/l BaA and 34.5 μg/l BaP.

Soils

Carcinogenic PAH settle on soil and are absorbed there by microorganisms or plants or decomposed by some bacteria. Some of the PAH found in soil and sediments may be synthesized by plants or organisms present in the soil.

Soil data are difficult to compare since information has been compiled by different researchers in different locations using different experimental techniques. In spite of this difficulty, some concentrations are listed here in Table 21 by compound and by type of contamination.

Table 20

PAH CONCENTRATIONS IN WATER

	µg/l Drinking Water	µg/l Surface Water
Benzo(a)pyrene	0.0001-0.023	0.0006-0.114
Chrysene	-	0.0118-0.038
Benz(a)anthracene	0.001-0.023	0.0043-0.185

Ref: IARC 1973

Table 21

CONCENTRATIONS OF PAH IN SOILS

(μg/kg)

	Benzo(a)pyrene	Chrysene	Benz(a)anthracene
Forest	Up to 1,300	—	5-20
Non-Industrial	0-127	—	—
Towns and Vicinities	0-939	—	—
Soil near Traffic	Up to 2,000	—	1,500
Near Oil Refinery	200,000	—	—
Near Airfield	785	—	—
Polluted by Coal Tar Pitch	650,000	600,000	2,500,000

Ref: IARC 1973

Note that soil directly contaminated by fossil fuel sources such as oil and coal-tar pitch tends to have concentrations several magnitudes greater than the other soils. Note also that a forest sample contained up to 1300 μg/kg, whichis almost as much as the levels found near traffic.

A study done in Russia found 83% of soil samples to contain less than 3 μg/kg of BaP. Podzolic soils had 0.7-0.8, soddy carbonate soils 11-13, Moscow city soils 269-347, and nearby Moscow freeway soils 16-67 μg/kg (Shabad 1971b). These values are quite a bit lower than those listed in Table 21.

Natural Sources

The sources of PAH that occur naturally in the environment were classified into three categories: plants, food (fresh and processed), and fossil fuels. In general, the benzo(a)pyrene content of dry organic substances is 10-20 μg/kg (Andelman 1970). BaP constitutes 1-20% of the total carcinogenic PAH. The environment surrounding these naturally occurring sources is sediments and soils that also contain PAH. As examples, consider ancient sediments of limestone and boghead, which contain 20, 40 μg/kg, respectively, of BaP. The origin of the PAH in these sediments is thought to be due to natural forces and plant and bacteria synthesis, and not due to any pollution (Mallet 1969). Consider also marine sediments, which contain BaP in the range of 1-5,000 μg/kg, depending on type and depth. These may indicate that certain marine organisms can concentrate and fix PAH (Andelman 1970, p. 487). The microorganisms in soils containing a high concentration of BaP--30,000 μg/kg--will tend to decompose 50-70% of the material. A lower concentration of BaP will not tend to be readily degraded in soils (Khesina 1969).

Plants

Plant seedlings were found to contain 10-20 μg/kg BaP of dried material after 8-10 days of growth in a PAH-free environment. Andelman (1970) also gave a general figure of 10-20 μg/kg BaP in active plant tissue. The amounts of BaA, BaP, and anthracene were estimated to be 5-110 μg/kg of dry plant material for each of 3 PAH (Hancock 1970). A German researcher found the BaP concentration of dried leaves to be 8-40 μg/kg (Graef 1966a). Various bacteria also concentrated BaP through synthesis in amounts of 2-10 μg/kg of dried material (Andelman 1970). Marine plants such as algae contained carcinogenic PAH in the range of 10-50 μg/kg. The BaP concentrations in tobacco leaves were determined to be 103 μg/kg and, after processing, 113 μg/kg (D'Arrigo 1972). Even higher concentrations for unsmoked tobacco of 54-270 μg/kg were found (Andelman 1970).

Foods

A lot of concern has surrounded the concentration of carcinogenic PAH in the food chain. The voluminous literature reflects efforts to determine the concentrations of these compounds in all fresh food groups and certain types of cooked and processed food. Most information was found for the compounds BaA, BaP, and chrysene. Most of the values reported were determined by different researchers and are, therefore, difficult to compare. Consider, firstly, the cereals and tubers food group in Table 22. The concentrations of the three PAH are pretty much the same. Note that the peelings in potatoes tend to have higher concentrations of PAH. Wheat and oat husks also have a great deal of BaP since the BaP concentrations of wheat and oats were found to decrease 60% and 40%, respectively, by simply removing the husks (Rohrlich 1971). Vegetable and fruit concentrations of PAH are in agreement with the general plant concentrations discussed under "Plants." Table 23 lists

Table 22

PAH CONCENTRATIONS IN CEREALS AND TUBERS

	Cereals µg/kg	Potatoes µg/kg	Ref.
Benz(a)anthracene	In general 0.4-6.8	-	IARC 1973
Chrysene	In general 0.8-14.5	-	IARC 1973
Benzo(a)pyrene	In general 0.25-0.84	Peelings 0.36 Tubers 0.09	Shabad 1972
	Barley, wheat, rye 0.2-4.1		Grimmer 1968

Table 23

PAH CONCENTRATIONS IN VEGETABLES AND FRUITS

(μg/kg)

	Cabbage	Kale	Spinach	Lettuce	Tomatoes	Other Fruits	Salad
Benz(a)-anthracene		43.6-230[a]	16.1[a]		0.3[a]		4.6-15.4[a]
Chrysene		58.5-395[a]	28.0[a]		0.5[a]		5.7-26.5[a]
Benzo(a)-pyrene	24.5[b]	12.6, 24.5[c]	7.4[c]	2.8-12.8[c]	0.22[c]	2-8[a]	
		12.6-48.1[a]	7.4[a]		0.2[a]		2.8-5.3

[a] IARC 1973

[b] Grimmer 1965

[c] Grimmer 1968

the BaP, BaA, and chrysene concentrations for selected vegetables and fruits. Note that leafy vegetables have quite a bit of PAH with kale having the highest concentration of each of the three PAH. Dairy products such as milk and butter were found to contain essentially no BaP (Grimmer 1968). Table 24 lists PAH concentrations in cooked and smoked meat and fish. Note that smoking the meat or fish increases the carcinogenic PAH content. This is probably due to pyrolytic synthesis of PAH during the smoking process (Wierzchowski 1972).

PAH concentrations in beverages such as teas, coffee, and whisky are listed in Table 25. It was found that fresh food concentrations were generally in agreement with plant concentrations as quoted under "Plants." Cooking, baking, and processing of food tends to increase PAH levels, as was seen for smoked meat and fish and oils and fats (see Table 24). Note the high concentrations of the individual PAH in coconut oil and fat.

Fossil Fuels and By-Products

Fossil fuels such as coal (IARC 1973) seem to have low BaP concentrations. Their by-products tend to have concentrations several magnitudes greater depending on the rate and temperature of processing. It is well known that pyrolysis of organics leads to the formation of PAH (IARC 1973; see also "Formation of PAH"). It follows, therefore, that products formed under high temperatures such as coal tar, coal tar pitch, petroleum asphalt, and creosote have unusually high concentrations of PAH. Table 26 gives a list of these concentrations of BaA, BaP, and chrysene.

A petroleum distillation product, such as hexane, was found to contain 280 and 23 μg/kg of BaA and BaP, respectively. Shale oil BaP was found to be amazingly low, 0.1 μg/kg, probably because shale oil processing causes the PAH to stay with the spent shale. PAH concentrations may also depend on processing--the TOSCO process is claimed to

Table 24

PAH CONCENTRATIONS IN
COOKED, SMOKED, AND PROCESSED FOODS
(μg/kg)

	Refined Oils or Fats	Fresh Fish Frozen or Salted	Broiled meat or Fish	Smoked Fish	Smoked Meat/ Sausages
Benzc(a)-pyrene	0.9-15[a]	< 0.1[b]	meat and sausages[a] 0.17-0.63	1.0-78.0[d]	0.02-107[a]
	margarine 0.2-6.8[a]			37[d]	
	coconut oil 43.7[a]		BBQ meat[a] 2.6-11.2	0.1-0.8[e]	
	coconut fat 62[a]		fish[a] 0.9	traces-2.1[a]	
			8.7-27.2[f]		
Chrysene	0.5-129[a]		ham[a] 0.5-2.6		
			fish 4.3[a]	0.3-173[a]	
			meat and sausages[a] 0.5-2.6		
			BBQ meat[a] 0.6-25.4		
Benz(a)-anthracene	0.5-13.5[a]		meat and sausages[a] 0.2-1.1		ham - up to 12[a]
	coconut oil 98[a]		charcoal broiled[a] 1.4-31		0.02-189[a]
	coconut fat 125[a]				

[a] IARC 1973
[b] Gorelova 1974
[c] Wierzchowski 1972
[d] Andelman 1970
[e] Grimmer 1968
[f] Shirotori 1972

Table 25

PAH CONCENTRATIONS IN BEVERAGES

(μg/l)

	Roasted Coffee	Teas	Whisky
Benzo(a)pyrene	0.3-0.5[a]	3.7-3.9[a]	0.04[b]
	0.1-4[b]	3.9-21.3[b]	
		Green teas[b] 0.5-16	
Chrysene	0.6-19.1[b]	4.6-6.3[b]	0.04-0.06[b]
Benz(a)anthracene	0.5-14.2[b]	—	0.04-0.08[b]

[a] Grimmer 1968

[b] IARC 1973

Table 26

FOSSIL FUEL AND ITS DERIVATIVES
(μg/g)

	Coal	Coal tar	Coal tar pitch	Petroleum asphalt	Creosote oil
Benzo(a)-pyrene	300-1000	30,000	12,500	0.1-27	0.00014-0.0002
Chrysene	—	Up to 2,860	Up to 10,000	Up to 0.4-34	Up to 1,340
Benz(a)-anthracene	—	Up to 6,980	Up to 12,500	Up to 35	Up to 2,940

Ref: IARC 1973

make more PAH than other alternatives. In comparison with coal tar, BaP concentration for wood tar was found to be only 0.34 μg/kg (Andelman 1971). Note the much larger concentration of BaP as compared with BaA in coal tar. Although carcinogenic concentrations in fossil fuels are not excessive, their derived products after processing under high temperatures accumulate dangerous amounts of PAH.

Anthropogenic Emissions and Effluents

PAH are formed under high-temperature pyrolysis of organic matter ("Formation of PAH"). The amount of BaP formed, for example, depends on how reducing the combustion atmosphere is. With increasing air-to-fuel ratios, BaP decreases in concentration (Lavrov 1972). PAH formation also seems to be associated with higher plants which contain more complex phenolic compounds such as lignin, but other types of organics can also produce PAH ("Formation of PAH"). Greater PAH formation rates are associated with coal combustion than with other fossil fuels. As evidence for this, the city of Budapest from 1965 to 1970 showed a decrease in BaP concentrations due to change from coal to oil (Kertesz-Saringer, 1972a). BaP in air is adsorbed on soot particles and is preferentially adsorbed on the smallest particles (Masek 1973). PAH are not soluble in water but exist also adsorbed on solid surfaces (McGinnes 1974).

Stationary

Power Plants--Table 27 gives a summary of the numbers found in the literature for heat generation. BaP is in considerably greater concentration than BaA in a coal-fired installation and emissions from a gas power plant tend to be much lower. It was not specified, for the stack gas emission reported, whether the power plant was oil, coal, or gas fired.

Table 27

HEAT GENERATION IN A COAL-FIRED INSTALLATION

PAH	Coal	Gas	Stack Gas
Benz(a)anthracene	19-3,900[a] $\mu g/10^6$ Btu	—	—
Benzo(a)pyrene	19-400,000[a] $\mu g/10^6$ Btu	20-200[a] $\mu g/10^6$ Btu	0.32 $mg/10^3 m^3$[b]

[a] IARC 1973

[b] Sawicki 1967

Industry--As expected, emissions and effluents from various industries are quite large, especially if they burn by-products of fossil fuels as fuels. Table 28 gives a summary of concentrations of three PAH from various industrial processes. While emissions from gas works are in a range comparable with those from coal power plants, note the excessive emissions from coal-tar pitch combustion. Note also that these emissions are more than 20-30 times those from coking plant ovens.

Other industries are also major sources of PAH. Foundries were found to emit 1-3 mg BaP/10^3 m^3 from the casting operations, depending on the temperature (Zdrazil 1965). An aluminum plant was found to emit 10 kg of BaP a day or 0.1 μg/sq. mile/day on the ground in the plant area (Olsen 1969). A fiberboard works pitch boiling plant was found to emit 1.2; on the premises, 0.2; at 100 meters, 0.1; and at 500 meters from the plant, 0.05 mg/10^3 m^3 (Bolotova 1967). A carborundum works crushing plant emission had 0.08, coke furnaces emission 0.06, and at 500 meters away from furnace 0.01 mg/10^3 m^3 of BaP. A vinyl phonograph records plant emitted 5.2 and a rubber products plant, depending on distance from source, 0.05-0.02 mg/10^3 m^3 BaP (Bolotova 1967).

In summary, high-temperature processes as found in gas works, coking and especially coal-tar pitch installations are heavy emitters of carcinogenic PAH. Other industrial processes using organic substances such as (poly) vinyl chloride and rubber can be expected to emit PAH to a lesser degree.

Incinerators--Emissions of BaP from various incinerators and open burning are summarized in Table 29. Municipal refuse incineration emits less BaP than outdoor burning. This is probably due to the more complete combustion in an incinerator.

Table 28

CONCENTRATIONS OF PAH FOR VARIOUS INDUSTRIAL PROCESSES ($\mu g/m^3$)

	Gas Works	Coal-Tar Pitch	Coking Plant
Benz(a)anthracene	0.8-14 (air from plant)[a]	0.7 (air)	—
		1,300 (ind. effluents)[a]	
Benzo(a)pyrene	0.18-7.3 (air from plant)[a]	0.4 (air)	159 (in ovens)
		2,700 (industrial effluents)	1.9 (@400 m)[b]
		6,000 (kettle @ 310°C, 20 cm from surface)[a]	1.2-40[c]
			1.3-92[d]
			0.1-1.6[e]
Chrysene	—	1,600 (industrial effluents)[a]	—

[a] IARC 1973

[b] Masek 1971

[c] Masek 1967

[d] Masek 1965

[e] Adamiak-Ziemba 1972

Table 29

BaP EMISSIONS FROM INCINERATORS AND OPEN BURNING

Type	$mg/10^3 m^3$
Garbage	1,400
Auto parts	170
Vegetable matter	14
Municipal refuse	2.6
Open burning (grass, leaves)	4.2

Ref: IARC 1973

Another comparison of the emissions in incinerators and of open burning for BaA and BaP was made (IARC 1973). Note that the concentrations are listed with respect to weight of particulate matter rather than volume in Table 30. This table confirms the differences between open burning and municipal refuse incineration.

Since PAH compounds are produced when hydrocarbons are pyrolyzed ("Formation of PAH"), internal combustion engines associated with automobiles, trucks, airplanes, and railroads should be a source of these compounds. Jet airplanes should also contribute PAH compounds to the environment. The amount and type produced should be a function of fuel, engine type, and engine duty. Under heavy loads or fuel-rich conditions where combustion is not complete, PAH generation rates will be higher than for light loads or fuel-lean conditions. The PAH compounds produced will probably be adsorbed on the particulates produced by combustion.

Table 31 shows some PAH levels associated with diesel and gasoline internal combustion engines. The measurements were made by different investigators using a different reporting basis so that it is not possible to draw inferences about the effect of fuel and engine type from these data.

Table 30

COMPARISON OF PAH LEVELS IN INCINERATION AND OPEN BURNING [a]

PAH	Municipal	Commercial	Open
	(mg/kg of particulate matter)		
Benz(a)anthracene	0.09-0.26	5-210	25-560
Benzo(a)pyrene	0.02-3.3	58-180	11-1100

[a] IARC 1973

Table 31

PAH IN EXHAUST GAS FROM
DIESEL AND GASOLINE ENGINES[a]

	Diesel	Gasoline
Benz(a)anthracene	2.3-15 $\mu g/m^3$ exhaust gas [a]	61.7 mg/kg exhaust gas (4.2 μg/minute) [a]
Benzo(a)pyrene		0.6-7.4 $\mu g/m^3$ exhaust gas [a] 31.5 mg/kg exhaust gas (2.2-9.6 μg/minute) [a]
Chrysene	3.6-17 $\mu g/m^3$ exhaust gas [a,b]	175 mg/kg exhaust gas (12 μg/minute) [a]

[a] IARC 1973

[b] Yakovlev 1975

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16. ABSTRACT

A review of the recent literature on polynuclear (polycyclic) aromatic hydrocarbons (PAH) has been carried out for general information on PAH and specific details about six selected PAH. The sources, transport, chemical and physical transformations, structure-reactivity relationships, and biological (non-carcinogenic) properties have been reviewed with recommendations for further research.

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